STUDY OF CLINICAL PROFILE AND RISK FACTORS IN ACUTE ISCHEMIC STROKE IN GOVERNMENT VELLORE MEDICAL COLLEGE HOSPITAL, VELLORE

A DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations for the award of the degree of

M.D. GENERAL MEDICINE – BRANCH I

DEPARTMENT OF GENERAL MEDICINE

GOVERNMENT VELLORE MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

APRIL 2016

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This is to certify that the dissertation titled **"STUDY OF CLINICAL PROFILE & RISK FACTORS IN ACUTE ISCHEMIC STROKE IN GOVERNMENT VELLORE MEDICAL COLLEGE HOSPITAL,VELLORE"** is a genuine work done by ***Dr.MAGUDEESWARAN R,*** Post Graduate student (2013 – 2016) in the Department of General Medicine, Government Vellore Medical College, Vellore under the guidance of **Prof.Dr.S.P.Kumaresan M.D.,DCH.,**

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Page count:	77
Word count:	9,878
Character count:	56,251
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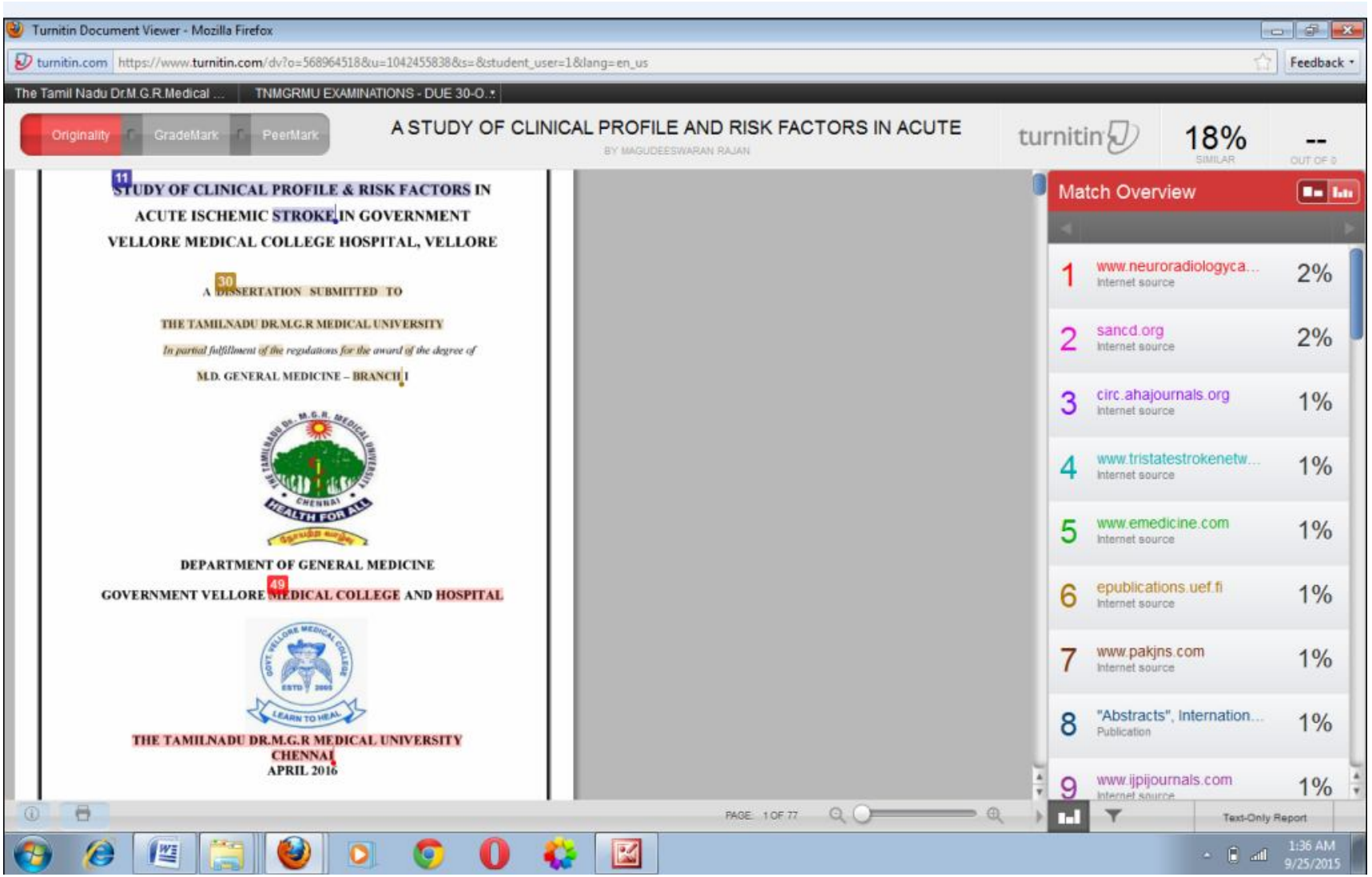
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This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the university regulations for the award of M.D., Degree in General Medicine(Branch – I)

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ACKNOWLEDGEMENT

It gives immense pleasure for me to thank everyone who has helped me during the course of my study and in preparing this dissertation.

My sincere thanks to **Dr.G.Selvarajan, M.S., DLO.,** the Dean, Govt. Vellore Medical College for permitting me to conduct the study and use the resources of the College.

I am very thankful to the chairman of Ethical Committee and members of Ethical Committee, Government Vellore Medical College and hospital for their guidance and help in getting the ethical clearance for this work.

I consider it a privilege to have done this study under the supervision of my beloved teacher, guide, and unit chief **Prof. Dr.S.P.KUMARESAN M.D., DCH,** who has been a source of constant inspiration and encouragement to accomplish this work.

I express my deepest sense of thankfulness to my Assistant Professors **Dr. B.GOWRISHANKAR M.D., DCH, Dr. G.ELUMALAI M.D., Dr. M. SUMATHIRA M.D.,** for their valuable inputs and constant encouragement without which this dissertation could not have been completed.

I express my sincere gratitude to **Prof.Dr. J. Philomena M.D.,** Professor &Head of the Department of General Medicine. I also thank **Prof. Dr.D.ANBARASU M.D.**, Professor of General Medicine, **Prof.Dr.Muthu chella kumar M.D.**, Professor of General Medicine and **Dr.G.O.Bharani M.D.,D.Diab**., Former professor & Head of the Department of General Medicine **Prof. Dr.Ravikumar MD ,DM**, Professor of Neurology and **Dr.T.Muthu MD, DM**, Asst. professor of Neurology for their valuable inputs and support.

I am particularly thankful to my fellow postgraduate colleagues **Dr. Arun Natesh R, Dr. Chandru J** and **Dr. Vijayakumar N** for their valuable support in the time of need throughout the study.

I thank my junior Post Graduate **Dr. Aswinth R** who supported me in completing the dissertation. I extend my sincere thanks to junior postgraduate **Dr.Ashwini metry & CRRIs** for helping me in the preparation of this dissertation

It is my earnest duty to thank my friends and family members without whom accomplishing this task would have been impossible.

I am extremely thankful to my patients who consented and participated to make this study possible.

ABBREVIATIONS

CNS	-	Central Nervous System
CAD	-	Coronary Artery Disease
UMN	-	Upper Motor Neuron
LMN	-	Lower Motor Neuron
DM	-	Diabetes Mellitus
AF	–	Atrial Fibrillation
TIA	-	Transient Ischemic Attack
CT	-	Computed Tomography
MRI	-	Magnetic Resonance Imaging
DWI	-	Diffusion Weighted Imaging
ECG	-	Electrocardiogram
FBS	–	Fasting Blood Sugar
PPBS	-	Post Prandial Blood Sugar
HBA1C	-	Glycated Haemoglobin
HDL	–	High Density Lipoprotein
LDL	–	Low Density Lipoprotein

VLDL	-	Very Low Density Lipoprotein
HMG CoA	-	Hydroxyl Beta Methyl Glutaryl Coenzyme A
APLA	-	Anti PhosphoLipid Antibody
ANCA	-	Anti Neutrophilic Cytoplasmic Antibody
OCP	-	Oral Contraceptive Pills
BMI	-	Body Mass Index
DALY	-	Disability Adjusted Life Year

ABSTRACT

BACKGROUND **:** Stroke or cerebrovascular accident is one of the leading cause of morbidity and mortality in adult life. Ischemic strokes are most common type of strokes and they account for 80%–85% of cerebrovascular accidents worldwide. Clinical presentation of stroke is variable depending upon the involvement of vascular territory. Etiology of ischemic stroke varies in different population due to differences in lifestyle,environmental and genetic factors. It also varies in different age groups. Hypertension, diabetes, dyslipidemia and smoking have been identified as major risk factors. Many of the risk factors for stroke are modifiable. Recognition and early intervention of risk factors can reduce the incidence of ischemic stroke.

OBJECTIVES:

To study the clinical profile & frequency of association of various risk factors in ischemic stroke patients at Govt. Vellore medical college, Vellore

MATERIAL AND METHOD

It is an Hospital based cross sectional study conducted over a period of one year among 100 ischemic stroke patients who were admitted in medical ward at Govt. Vellore medical college hospital, Vellore.

RESULTS: The Ischemic stroke incidence is high in the age group of 56-70 years. It is seen that 78% of the sufferers were in the age group >45

years . Stroke is more common in males than females (ratio 3:1).Most of the stroke patients (66%) in our study belong to low income group. Most common clinical presentation was hemiplegia/hemiparesis(88%). Cranial nerve involvement is noted in 67% patients and Speech disturbances are found in 44% patients. Most common vascular territory involved is middle cerebral artery. Around 92% of patients had middle cerebral artery infarction. **In our study hypertension, smoking, alcoholism, dyslipidemia were significantly associated with stroke in patients with age group more than 45 years.** In 100 total stroke patients 64% had hypertension and it is found to be high when compared to previous studies. Among 100 ischemic stroke patients 16% of patients had both Hypertension and Diabetes. Smoking and alcoholism were identified only in male population. In total stroke patients, 36% of patients were smokers & 35% were found to be alcoholic.10% of patients had past episode of transient ischemic attack or stroke. 15% of patients had heart disease or atrial fibrillation.

Conclusion:

Ischemic stroke has a male predominance and hypertension was found to be a most common risk factor. Systemic hypertension, smoking, alcoholism, dyslipidemia were significantly associated with stroke in patients with age more than 45 years.

Key words: Ischemic stroke, clinical profile, risk factors

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INTRODUCTION

STROKE is the **second commonest cause of death**. It is the fourth leading cause of disability world-wide. Nearly twenty million people each year will suffer from stroke and of those five million will die. Death due to stroke is no longer in developed world. 85.5% of total stroke death occurs in developing countries.[1] The morbidity of stroke in developing countries was approximately seven times that in developed countries.

Recent studies identified 0.9 – 4.5% of medical and 9.2 – 30% of neurological admissions in India were due to cerebrovascular accident. The **case fatality rate of stroke** during the time of hospital discharge is **9%. At the end of one month, fatality rate increases up to 20%.[2]**

A stroke is caused by loss of the blood supply to the brain. This cuts off the oxygen and glucose causing irreversible damage to the tissues of brain parenchyma. **WHO** clinically defines stroke as "***the rapid development of clinical signs and symptoms of a focal neurological disturbance lasting more than 24 hours or leading to death with no apparent cause other than vascular origin***".

Ischemic stroke is caused by loss of blood supply to brain tissues due to sudden occlusion of arterial system . The occlusion may be due to emboli or thrombus. Ischemic stroke is responsible for 50 – 85% of all strokes world-wide.[3]

Non modifiable risk factors for stroke include age, sex and genetic factors. The **modifiable** risk factors for ischemic stroke include systemic hypertension, alcoholism, smoking, hyperlipidaemia, diabetes etc.

By targeting various modifiable risk factors we can reduce the incidence of stroke. Study of Non-modifiable risk factors also helps in identifying high risk population.

Distribution of risk factors and their influence on ischemic stroke may be variable depending upon the geographical area and cultural background. Our study focused on clinical profile and various risk factors that influencing the occurrence of ischemic stroke in and around the Vellore area.

REVIEW OF LITERATURE

Stroke or a cerebrovascular accident is a clinical syndrome characterized by loss of cerebral function due to tissue hypoxia. The **WHO** clinically defines stroke as "Rapid development of clinical signs and symptoms of a focal neurological disturbance lasting more than 24 hours or leading to death with no apparent cause other than vascular origin".[4,5] When the focal neurological deficits last only for a short period of time (usually less than an hour), this is called a transient ischemic attack (TIA). It is the second most common cause of death in adult population. Stroke has been divided into two types based on its pathophysiology:

Ischemic strokes –Ischemic strokes are most common type of strokes and they are caused by either cerebral thrombosis or embolism. They account for 80%–85% of cerebrovascular accidents worldwide.[3] One of the most common causes leading to ischemic stroke is narrowing of blood vessels of head or neck. Narrowing of vessels are mainly due to atherosclerosis and cholesterol deposition. As narrowing of vessels progresses, stasis of blood occurs, this in turn leads to formation of blood clots**.** These blood clots can occlude the vessels at the site of formation (thrombosis) or they can dislodge and become trapped in distal site (embolism) and cause ischemic injury to brain tissues**.** Another cause of stroke is cardio embolism, which can occur as a result of thrombus formation inside the heart chambers. The conditions like atrial fibrillation, myocardial infarction, valvular lesions, or cardiomyopathies can

influence the formation of thrombus inside the heart chambers. There are some minor causes for ischemic stroke include traumatic injury to the blood vessels of the neck, and disorders of coagulation.

Thrombotic stroke: A form of stroke, where the underlying pathology is formation of thrombus leading to occlusion of blood vessels in cerebral circulation. Almost 50% of all stroke cases belong to this type of thrombosis to blood vessels. Thrombosis in cerebral circulation occurs broadly in two type blood vessels- large vessels and small vessels. Thromboses involving large blood vessels like anterior cerebral artery, middle cerebral artery and posterior cerebral artery which causes infarct in large areas correspondingly. Thrombosis in small penetrating arteries causes lacunar infarct in different parts of brain.

In **Embolic Stroke**, blood clot from other parts of the body reaches blood vessels of brain. Mostly blood clot arises from the cardiac source or from carotid artery and these clot (emboli) passes through the cerebral circulation. Within the cerebral circulation, at the site where the artery narrows or bifurcates, these clot gets lodged and disrupts blood flow to brain parenchyma causing stroke in the form of focal deficits or transient ischemic attack.

Haemorrhagic Strokes- Haemorrhagic stroke may be due to subarachnoid hemorrhage or intracerebral hemorrhage. Sub arachnoid hemorrhage accounts for 1%-7% of all strokes and intracerebral hemorrhage constitutes 7%-27% of all strokes worldwide.[3]

Stroke is a heterogeneous syndrome which may be due to any disease process that disrupts the cerebral circulation and resulting in subsequent hypoxic tissue damage. The manifestation of a stroke depends on anatomical location and severity of tissue damage. It may cause sudden motor weakness, sensory disturbances, slurred or complete loss of speech, vision or gait abnormalities. Since cerebral cortex has different areas and functions, it is usually the area supplied by particular vascular territory that is affected. The warning signs of stroke should be immediately recognised for early medical intervention.

WHO world health report-2007 states that-[6]

* In each year approximately 15 million people suffer stroke worldwide. Of these, one third die and another one third are permanently disabled.
* Uncontrolled hypertension contributes to over 12.7 million strokes worldwide.
* In developed countries, the incidence of stroke is declining –It is mainly due to efforts to lower blood pressure and less smoking. However, the overall rate of stroke remains high due to the aging of the population.
* In India, Stroke is potentially the most devastating consequence of vascular disease, causing serious long-term disability and incurring extremely high medical, emotional and financial costs.

Pathophysiology

Stroke is a "sudden neurological event with sudden or rapid progression of signs and symptoms involving specific areas of brain".[7] In stroke caused by

ischemia, blood supply to the brain is blocked and brain tissue is deprived of the glucose and oxygen. Ischemic stroke is caused by multiple causes and has subsequent sequelae. Thrombus can form in arteries from both outside and inside the cranium, when the roughened intima is colonised by plaque. When the endothelial injury has occurred coagulation cascade is activated and thrombus forms at the time of plague formation. Collateral system takes up and maintains the function. When the collateral system is also compromised eventually cell death occurs and the condition worsens.

In **embolic stroke,** clot blocks the cerebral vessels from a different source. Micro embolus has many sources such as from cardiac origin like patent foramen ovale, atrial fibrillation and infective endocarditis. 20% of ischemic strokes are cardioembolic in origin.[8] The source of emboli can be in any form like blood, fat, air occurring commonly during surgical procedures. Other less common causes are aortic dissection and coagulopathies, arteritis, infection, and drug abuse such like cocaine.[9,10] Thrombus or emboli decrease the blood supply to the brain and lead to ischemic cascade. The optimal balance of temperature, PH, waste removal and nutrition or required for the brain for their optimal function. Scientific research during the last three decades reveals environmental alterations involved in the pathophysiology of ischemic stroke. Understanding the ischemic cascade and injury at cellular level has led to the concept of a **therapeutic time window** for the purpose of early intervention. The dead cells are surrounded by an area of hypo perfused region known as penumbra region.[11] Pharmacological and non-pharmacological treatments are

used to stop the cellular events in ischemic cascade.

Risk Factors

Careful understanding of risk factors can help in avoiding the onset of stroke. Age, gender, race, ethnicity, hereditary are non-modifiable risk factors. Systemic Hypertension, smoking, chronic alcoholism, dyslipidemia, obesity, sedentary life style, atrial fibrillation, previous heart disease, transient ischemic attack are potentially treatable/modifiable factors in the incidence of stroke. The epidemiological studies on stroke are much least in developing countries. The prevalence of stroke is 40-270 in rural populations which is lower compared to western countries.[12] The reason for variation may be due to socioeconomic factors, ethnicity & cultural habits.

Non Modifiable Risk Factors

The understanding of non-modifiable risk factors help in prevention or treatment of the patients at the high risk.

Age

The progressive nature of both modifiable and non-modifiable risk factors with respect to increase in age contributes to the development of stroke. With risk factors the incidence of stroke doubles beyond 55 years of age.[13]

Sex

Stroke is more common in males than in females and the incidence rate is also high in men.[14,15] In age group between 35-44 and in age group above 85, the stroke rate is slightly higher in women than men. The rate of stroke among

female in the year 1997 accounted for about 60.8%. Thus 1 in 6 patients die among the female population due to stroke.[16] Risk factors such as pregnancy and the use of OCPs contribute to the incidence of stroke among women.[17]

Family history

Maternal and paternal history of stroke is also a risk factor for stroke.[18] The mechanism by which it contributes to stroke includes inherited risk factors of stroke, cultural & religious background in families.[19] Twin studies suggest the strong inheritance of stroke in that too the rate is more among monozygotic twins than in dizygotic twins.[20] There is fivefold increase in stroke rate in monozygotic twins.[21]

Modifiable Risk Factors

Modifiable risk factors are as follows:

Hypertension

Hypertension contributes to both cerebral infarction and cerebral haemorrhage.[22,23] Hypertension, is the single most important modifiable risk factor for cerebrovascular accident. It causes about one half of ischemic strokes and also increases the risk of hemorrhagic stroke. Vascular remodelling caused by systemic arterial hypertension is the firsts step in the development of atherosclerosis and lipohyalinosis. Increase in either systolic or diastolic blood pressure or, both can precipitate stroke. Systolic pressure increases with age.[24] Increase in systolic blood pressure alone can contribute to stroke irrespective of the diastolic blood pressure.[25] A meta-analysis of 18 long-term randomized trials found that both ß- blocker and high-dose diuretics were effective in

preventing stroke.[26,]

Smoking

Smoking is a major risk factor for the development of stroke. It affects by involving both the vasculature and the blood component. In smokers the elasticity of the vessel is altered. The compliance of the vessel is reduced. Smoking causes increased arterial wall stiffness.[27] It increases viscosity of blood by increasing hematocrit. It damages the endothelial lining of blood vessels which promotes thrombosis. It also activates platelet aggregation. It activates the coagulation cascade by several mechanisms. Smoking is associated with several abnormalities which may contribute to thrombosis. Many smokers had increased fibrinogen levels and decreased HDL cholesterol levels.[28]

A meta-analysis of 22 studies shows an approximate doubling of the relative risk of ischemic stroke among smokers versus non-smokers.[29] In Framingham Heart Study there is 1.8-fold increase in stroke risk associated with smoking (after control for other stroke risk factors).[30] The prevention of initiation of smoking in former smokers plays an important role in the prevention of stroke. The risk of stroke decreases with time since the individual has stopped smoking. The Framingham Heart Study found stroke risk after cessation of smoking reduced to the level of non-smokers at 5 years from cessation.[31]

Exposure to atmospheric tobacco has a role in the incidence of stroke. Because of increased exposure to environmental tobacco even a small increase

in other risk factors may contribute to the development of stroke. Smoking contributes to atherosclerosis which is the important cause of both stroke and coronary artery disease. After controlling of other co factors Bonita and colleagues found a 1.82-fold increase in the risk of stroke among non-smokers who are exposed to atmospheric tobacco.[32] In summary, individuals exposed to environmental smoke has an equal rate of risk compared to active smokers. (risk of stroke is 18% for active smokers, 6% for non-smokers, 12% for individuals exposed to environmental tobacco)

Diabetes mellitus and metabolic syndrome

Diabetes and insulin resistance can lead to atherosclerosis and development of other risk factors including hypertension, obesity and abnormal dyslipidemia. These risk factors constitutes metabolic syndrome.[33,34] The main components of metabolic syndrome are hyperinsulinemia and insulin resistance. Many case-control and prospective epidemiological studies have confirmed an independent effect of diabetes on the occurrence of ischemic stroke, with an increased relative risk in diabetics ranging from 1.8 to nearly 6-fold. In the Framingham Heart Study, even though, the diabetes much affects peripheral arterial system, coronary as well as cerebral circulation also affected. Finally in a study it was concluded that persons with diabetes have double the risk of ischemic stroke compared with non-diabetics.[35]

Hypertension is common in 40-60% of individuals with type 2 diabetes. The frequency of complication of diabetes increases when the individual presents with both hypertension and hyperglycaemia. From various studies,

tight control of blood pressure in diabetic population significantly reduces the incidence of ischemic stroke.[36]

Atrial Fibrillation

Atrial fibrillation is an important cause of embolic stroke. In patients with non-valvular atrial fibrillation, 3% to 5% of patients had annual stroke incidence. Atrial fibrillation responsible for almost 50% of thromboembolic stroke.[37] The cause of stroke is cardioembolism in two third of patients with atrial fibrillation. The median age is 75 years. The Framingham Heart Study showed that there is well documented increase in stroke incidence in AF patients with respect to age. The risk of stroke increases from 1.5% for those 50 to 59 years of age to 23.5% for those 80 to 89 years of age.[38] Limited predictors attributing to the stroke risk has been documented from the randomised control trials for the development of atrial fibrillation. Old age, previous episodes of TIA or stroke, hypertension, left ventricular dysfunction, diabetes mellitus and female population with age more then 75 years are main risk factors of AF.[39] Randomized control trials showed Long-term oral anticoagulation of high risk patients will reduce the ischemic stroke risk by 68%.[40]

Other cardiac disease

Other cardiac causes of stroke that lead to thromboembolism are rheumatic heart disease with valvular lesions, prosthetic cardiac valves, dilated cardiomyopathy and congenital heart diseases with shunt lesions. Thus 20% of ischemic stroke is caused by cardioembolism.[41] The development of CVA is strongly associated with both symptomatic[42,43] and asymptomatic[44] disease.

Farmington study states that 8% of male patients with coronary artery disease will develop stroke in next six years. In female patients with myocardial infarction the incidence of stroke is 11%. Myocardial infarction can also lead to atrial fibrillation which causes cardioembolism leading to stroke. However majority of the strokes occurring due to myocardial infarction are due to ischemia.[45]

Sickle cell anaemia and stroke

Sickle cell anaemia is an autosomal dominant disorder with wide clinical manifestations. It may present with haemolytic anaemia with symptoms presenting with painful extremities and bones, bacterial infections and end organ infarctions. It also presents with impaired growth and cognitive dysfunction. Stroke is more common in homozygous ss disease. It is the common cause of young stroke affecting individuals of age group 20 by 11%.[46] The prevalence of stroke with sickle cell anemia is common in early childhood. Early detection by transcranial Doppler in sickle cell disease patients helps in the primary prevention.The rate of developing stroke is 1% in sickle cell disease patients but individuals with evidence of high blood flow velocity by transcranial Doppler have rate of 10% of developing stroke.[47]

Hyperlipidaemia

Serum lipid abnormalities are mainly associated with coronary heart disease but also it influences the incidence of stroke.[48] However lipid lowering drugs can help reducing the stroke risk and carotid atherosclerosis as per recent study. Few studies on ischemic stroke reported a weak association between

serum cholesterol level and increased incidence of ischemic stroke.[49] For instance men with high cholesterol level have increased mortality rate.[50] For individuals with serum cholesterol 240 to 279 mg/dL, the risk ratio is 1.8 and with cholesterol levels ≥280 mg/dL, the ratio is 2.6.[51] Recently using ultrasound technology they found relation between lipid levels, intimal plaque thickness and extracranial carotid atherosclerosis.[51,52] Older studies does not show much relationship between serum cholesterol and lipid lowering drugs.[53] More recent studies with HMG CoA reductase inhibitors have proved the reduction of level of LDL and thus gradual development of asymptomatic atherosclerosis.

In patients with coronary artery disease taking statins, the prevention of stroke has been proved by several meta-analysis.[53] The mechanism by which the statins work on stroke is not clear. Known other mechanism of actions by which it helps in the prevention of stoke other than lowering of lipoproteins is by stabilising the plaque, antithrombotic, anti-inflammatory and neuro protective mechanisms.[54]

Other modifiable risk factors

Obesity

Body mass index >30 kg/m acts as a predisposing factor for both coronary artery disease and stroke. Obesity increases as the age advances and is also associated with increase in blood pressure, blood sugar, dyslipidemia. These factors increases stroke risk in obese individuals. However several studies have proved relationship between abdominal obesity rather than BMI.[55]

In females,there is an increased risk for ischemic stroke with increased BMI. The relative risk ranged from 1.7 for BMI of 27 to 28.9 kg/m^2, 1.90 for BMI of 29 to 31.9 kg/m^2 and 2.37 for BMI of >31.90 kg/m^2. Increase in weight in age group >18years is also related with the increasing risk of developing stroke as the age advances.[56]

Physical inactivity

Physical activity has a vital role in preventing metabolic complications. The premature death and cardiovascular disease can be prevented by regular physical activity. Stroke prevention can also be made by regular physical activity.[57] Many studies like, Honolulu Heart Program, Oslo Study and Framingham Heart Study have also shown the protective effect of physical activity on men.[57,58] According to Copenhagen city heart study and Nurses health study, there is an inverse relationship with the level of physical activity and the incidence of stroke.[58] It is documented that leisure time physical activity reduces the incidence of stroke.[59]

The protective effect of physical activity is mainly by its role in controlling or modifying various known risk factors for ischemic stroke such as systemic hypertension, obesity, glucose intolerance and reductions in plasma fibrinogen & platelet activation. In other way physical activity improves HDL concentrations and plasma tissue plasminogen activator activity.[60]

National institute of health recommends moderate exercise for at least 30 minutes per day on all days of the week. Mild to moderate activity reduces the incidence of stroke to a greater extent and further reduces as the level of

recreational activity increases. Thus physical activity works in a greater extent in the prevention of stroke.

Alcohol abuse

The influence of alcohol on the occurrence of stroke is mainly dose dependent and controversial. For haemorrhagic stroke there is a direct relationship based on the amount of alcohol consumed.[62] Chronic drinking and acute intoxication of alcohol leads to cerebral infarction in young adults.

Studies have reported a J shaped curve between the alcohol consumption and the ischemic stroke. Having a protective effect on individuals taking 2 drinks per day and increased risk on persons taking more than 5 drinks per day compared with non-drinkers. The mechanism by which the alcohol consumption produces stroke is by increasing blood pressure, hyper coagulable states and by producing various arrhythmias. Mild to moderate drinking of alcohol reduces the risk of coronary heart disease, increases the serum HDL levels although it cannot be advised to take moderate among non-drinkers, it is advisable for chronic drinkers to take moderate amount thus helping in reducing the incidence of stroke.

Hyperhomocysteinemia

Many case control studies have showed relationship between the levels of homocystiene and the development of stroke. Although more case control studies have showed relationship between them much of confirmatory findings are required.[63] Elevated homocystiene levels in the body can be reduced by using folic acid, vitamin B6 and B12, but no trials have confirmed that

reducing homocystiene levels will help in reducing the incidence of stroke.[64]

Drug Abuse

Drug abuse increases the incidence of both ischemic and haemorrhagic stroke. The association between drug abuse and stroke risk is observed in a study conducted on lower socioeconomic population.[65] There is a convincing evidence that there is a seven fold increase in the incidence of stroke among drug users.[65] The mechanism by which it produces stroke is by increasing the blood pressure, haematological and haemostatic abnormalities that leads to increased blood viscosity and vasculitis.

Hypercoagulability

Blood disorders are responsible for 5 to 10% of ischemic stroke with an increased frequency in younger patients. Mutations in Factor V Leiden (having resistance to activated protein C), deficiencies in Protein C, Protein S &antithrombin III, and disorders of abnormal platelet function are the major hematological conditions that may lead to cerebrovascular events. Several case control studies have confirmed the relationship between the antiphospholipid antibodies (ApL) and the development of ischemic stroke. Presence of antiphospholipid antibodies can be detected by measurement of lupus anticoagulants (less specific) and anticardiolipin antibodies (more specific). Prolonged anticoagulation therapy in these groups of people may help the primary prevention of stroke. More studies are required to assess the benefits of lifelong anticoagulation.

Oral contraceptive use

The stroke risk with use of OCPs are mainly dose related.[66] Studies with second generation OCP's containing low levels of oestrogens are associated with decreased risk compared to first generation OCP's.[67] Although many studies reported an increased risk, one study reported a little association between the development of stroke and the use of first, second, third generation oral contraceptive pills.[68] Women having other risk factors other than use of OCP's like cigarette smoking, hypertension, diabetes have migraine are at an increased risk of developing stroke.[69]

Global Stroke Morbidity and Mortality

According to the World Health Organization, 15 million people suffer stroke worldwide each year. Of these, 5 million die and another 5 million are permanently disabled. High blood pressure contributes to more than 12.7 million strokes worldwide. In developed countries, the incidence of stroke is declining, largely due to efforts to lower blood pressure and reduce smoking. However, the overall rate of stroke remains high due to the aging of the population.

Source: World Health Report – 2002, from the World Health Organization 15 million new acute strokes every year.

28,500,000 DALYs (disability adjusted life-year) 28-30 day case fatality ranges from 17%-35%.

Stroke Morbidity and Mortality in India

Prevalence of stroke in India is 55.6 per 100,000 all ages and mortality

is 0.63 million deaths.

1.44-1.64 million cases of new acute strokes every year 6,398,000 DALYs.

12% of strokes occur in the population aged <40 years 28-30 day case fatality ranges from 18-41%.[70]

The distribution, prevalence and incidence of stroke in India

Inadequate morbidity and mortality rates are available in India due to improper death certification, incorrect death classification, or due to unknown aetiologies. Although a system of registration of death was introduced in India, from the year 1998, only 14% of the death is recorded accordingly to the causes.[70] Based on these registrations, 24% of the deaths comes under the death due to the circulatory systems (comprising of stroke) between the year 1998-1999 (in India the prevalence of stroke is more in urban areas than in rural areas.[71,72] Estimates for time trend for stroke are not available in India. The estimated mortality rate due to stroke was 8% in the year 1998-2004.[73] The incidence rate of stroke is about 1.1% in high income countries and 5.5% in low to middle income countries. The Indian National Commission on Macro-economics and Health found that the number of strokes will increase from 1,081,480 in 2000 to 1,667,372 in 2015.[73]

Prevalence/incidence of stroke in India

The stroke prevalence is limited and suffers from frequent bias and are not consistent due to improper diagnostic criteria. In India the crude prevalence rate varies according to the region. The rate is higher in urban than rural areas.

The Parsi population have the increased risk of stroke in India, since they migrated from Iran in the 7th century. In India the prevalence of stroke is estimated to be between 84-262/100,000 in rural and between 334-424/100,000 in urban areas. Although the individual studies have varying results, the global burden of study, estimated to be 89/100,000 in 2005 which is expected to increase from 91/100,000 in 2015 and to 98/100,000 in 2030.

Overall burden of stroke in India

As the prevalence of stroke is declining in developing countries ,India is still facing the epidemicity of stroke as the incidence increases, India will face the cost related problems concerning to stroke. It was recorded that 0.3%-9.4% of the total medical admissions are stroke and 9.5% to 30% of the total neurological case were stroke. It is found from the recent studies that 4% of all the incidence of stroke are under the age group of 40 years. Aged persons have a higher risk of stroke if they already had a previous attack. The amount spend in the treatment of stroke has not been much accurately calculated annually. Although the economic burden is not much known, 8.7 billion dollars is spend on coronary artery disease, stroke and diabetes. The number of death was 5,289,357 in 2004 which increased from 4,818,740 in the year 1998.

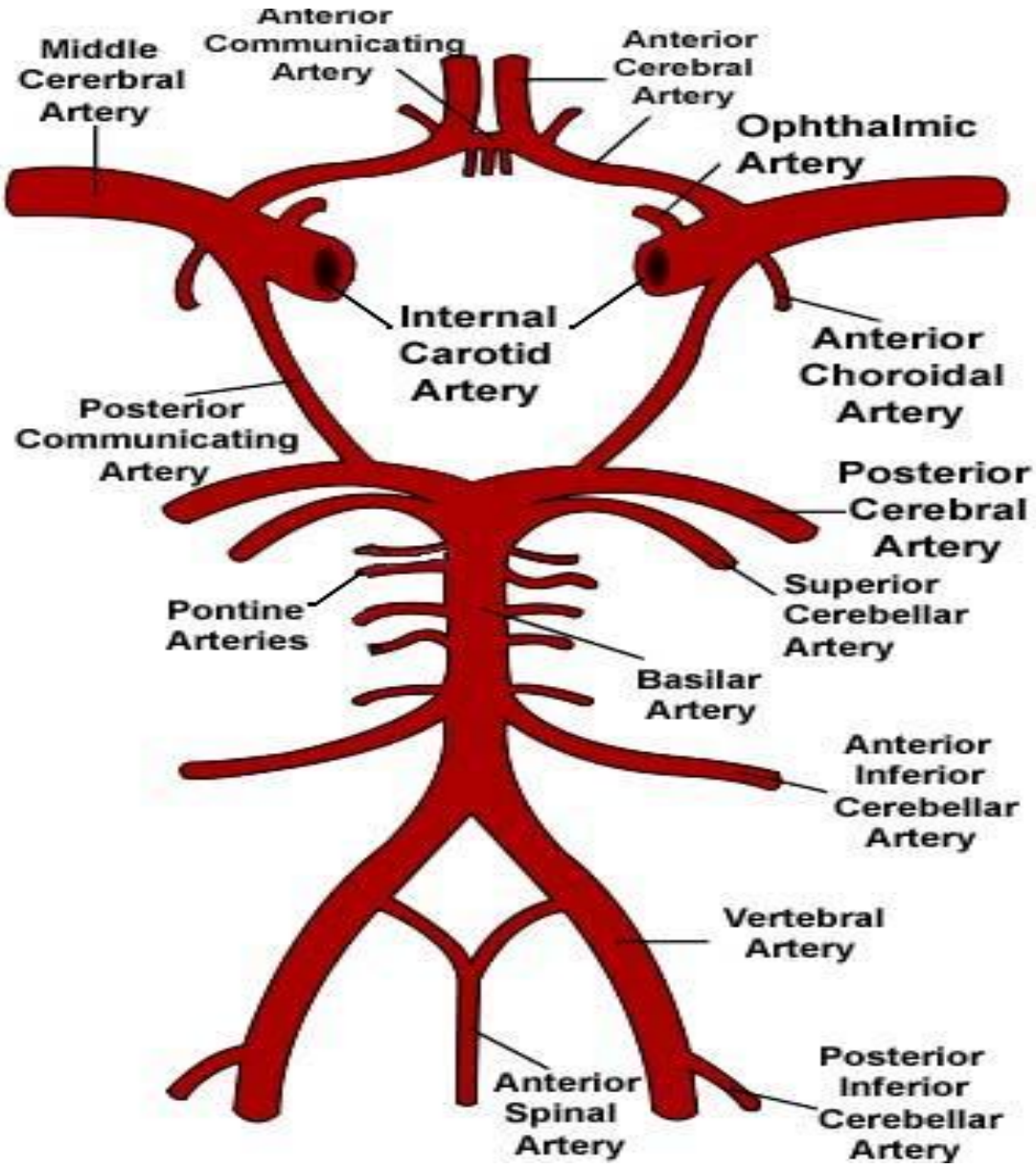
Stroke increases with age: The prevalence rate of stroke based on the individual studies from the Indian population is estimated to be varying 21/100,000 for the 20-40 age group to 625/100,000 in 60 and above group. The incidence rate increases from 27-34/100,000 in the 35-44 age group to 822-1116/100,000 in the 75+ age group.[73] In developing country like India, the

incidence of stroke is high in younger individuals compared to high income countries. The incidence of stroke varies from 18-32% among the younger individuals.

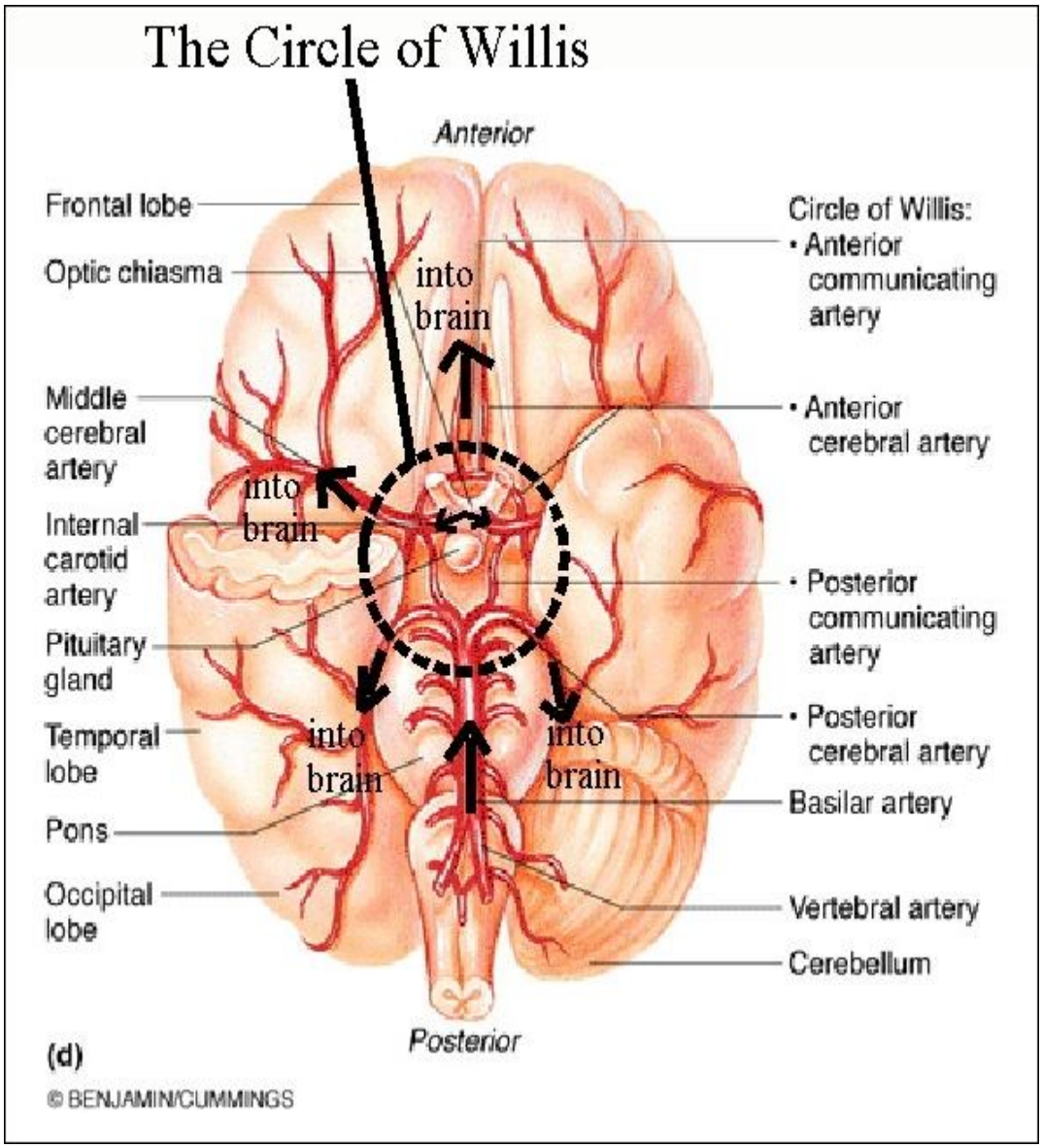
Male-Female ratio : The male to female sex ratio for the incidence of stroke is 7:1.[72] This is due to increased risk factors which are common in men than women like smoking and drinking. The mean onset of incidence of stroke ranges from 63-65 for men and 57-68 for women. Low socio economic status is associated with the higher rate for development of stroke due the prevalence of risk factors like high blood pressure, smoking, heavy alcohol drinking, heavy diet consumption.[74,75] Though the incidence of cardiovascular disease is more common among the high socioeconomic class, there is no evidence for the same for stroke.

Tripathi et al found out that the prevalence of risk factors is 7-10% greater in the low income population. Also the prevalence of stroke is more among the persons who had less than ten years of education than the persons who had more than sixteen years of education in India, around 24% and 54% respectively.[75]

BLOOD SUPPLY OF BRAIN



CIRCLE OF WILLIS


The Circle of Willis
Anterior
Frontal lobe
Optic chiasma
Middle cerebral artery
Internal carotid artery
Pituitary gland
Temporal lobe
Pons
Occipital lobe
into brain
into brain
into brain
into brain
Circle of Willis:
• Anterior communicating artery
• Anterior cerebral artery
• Posterior communicating artery
• Posterior cerebral artery
Basilar artery
Vertebral artery
Cerebellum
Posterior
(d)
© BENJAMIN/CUMMINGS

CLINICAL FEATURES OF STROKE

Any patient presenting with focal deficit or altered sensorium should be suspected and evaluated for stroke.

Sudden onset of any of the following clinical feature may be due to cerebral ischemia.

- Hemiparesis/hemiplegia, monoparesis/monoplegia, or (rarely) quadriparesis
- Hemi sensory defects
- Loss of vision (Monocular or binocular)
- Visual field deficits
- Facial weakness
- Double vision
- Aphasia (sensory or motor)
- Dysarthria
- Ataxia
- Vertigo (usually associated with any other feature)
- Altered level of consciousness

Clinical features can occur alone or combined with two or more features. Establishing the time at which the patient was last seen without stroke symptoms, or last known to be normal, is especially critical when fibrinolytic therapy is an option. There are many factors which are responsible for delay in

reaching hospital. Cerebrovascular accident that occurred in sleep may go unnoticed by caregiver or patient.

If patient noticed symptoms, on getting up from bed, then the time of onset of stroke is defined as the time at which he was last seen to be free of his current symptoms.

The goals of the physical examination are as follows:

- Look for aetiology - any evidence of atherosclerosis cardio embolism, carotid artery stenosis, atrial fibrillation, valvular lesions, shunt lesions, palpation of carotid, auscultation for carotid bruit etc
- Differentiate true stroke from stroke mimics
- Present degree of deficit to be documented for the purpose of assessment of disease progression
- Localization of the lesion anatomically
- Identification of associated co morbid conditions that influences outcome.
- Identification of conditions that altering treatment plan and decision (e.g, head trauma, coagulopathy, bleeding disorders, sepsis)
- Signs of meningeal irritation.
- Fundus - (for papilledema, micro aneurysm, emboli, cotton wool spots)

The physical examination should include all vital functions, starting with airway, breathing, and circulation. Patients with altered sensorium should be assessed for the need of intubation & airway protection. Massive infarcts &infarcts involving brainstem, can cause problems with airway patency, respiratory depression and cardiovascular collapse.

Head and neck, cardiac, and extremities examination

Any contusions, lacerations, and deformities on head may suggest traumatic head injury as the etiology. Carotid Auscultation may elicit a bruit, suggesting carotid artery stenosis as the cause of the cerebrovascular accident.

Cardio vascular examination may reveal underlying cardiac arrhythmias, valvular heart disease, congenital heart disease and any acute events like myocardial infarction or heart failure.

Peripheral arterial examination may detect unequal pulses or blood pressures in the extremities which may reflect the presence of aortic dissections, a rare case of ischemic cerebrovascular events.

Neurologic examination

Essential components of the neurologic examination include the following evaluations:

- Cranial nerves
- Motor system

- Sensory system
- Cerebellar functions
- Gait
- Deep tendon reflexes
- Language
- Mental status examination
- signs of meningeal irritation

National Institutes of Health Stroke Scale (NIHSS)

The NIHSS helps in assessing the severity and localization of the stroke. NIHSS scores help identification of patients suitable for fibrinolytic therapy.

NIHSS scale includes-

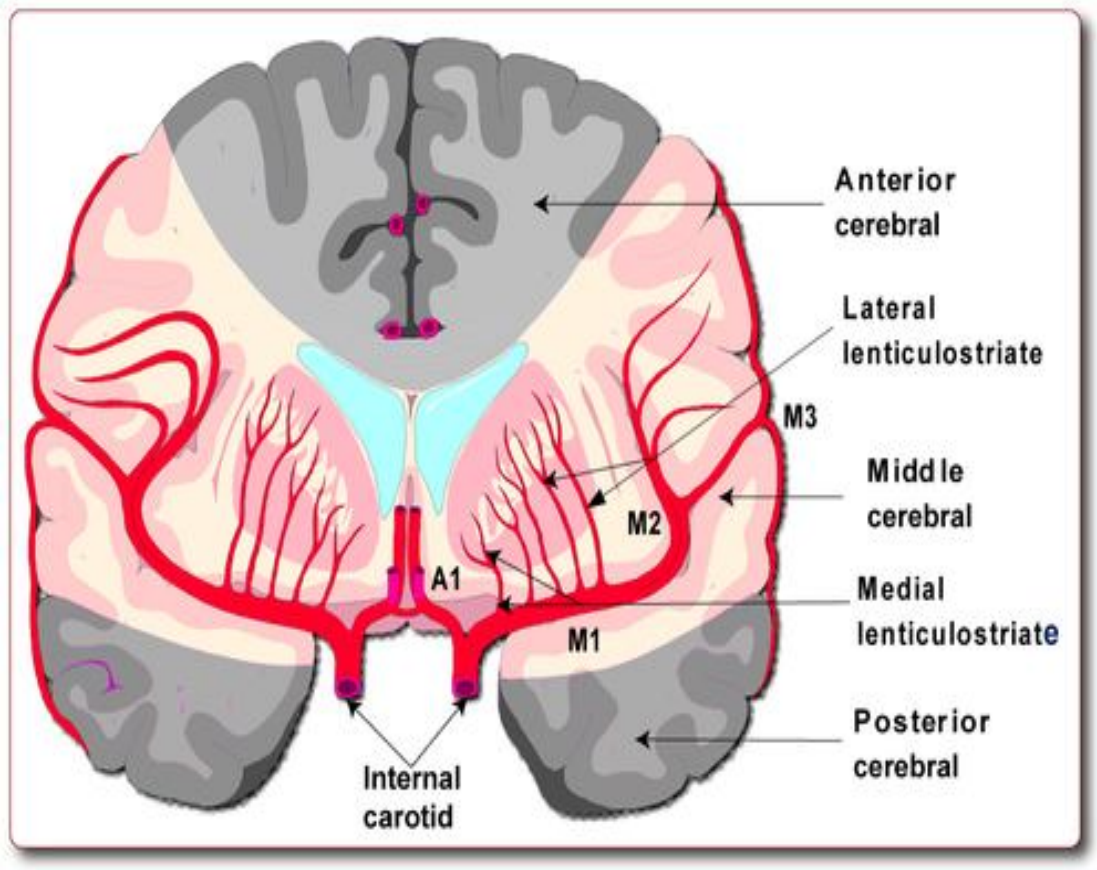
- level of consciousness
- Visual
- Motor
- Sensation and neglect
- Cerebellar function
- Language

Total score is 42. Score of less than 5 indicates minor stroke. A NIHSS score of >10 correlates with an 80% likelihood of proximal vessel occlusions.

Disadvantage:

The scale does not include some neurological deficits in PCA infarcts. (e.g. vertigo, ataxia)

MCA infarction:



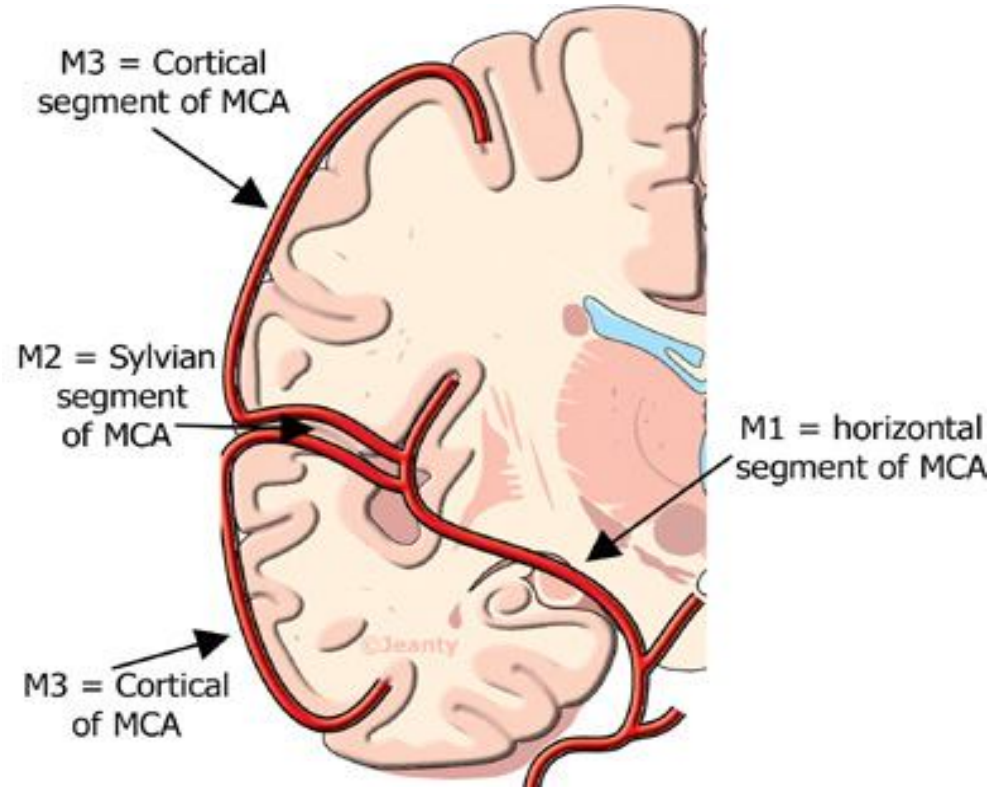
The internal carotid artery has two terminal branches.

A)Middle cerebral artery(MCA)

B)Anterior cerebral artery

The MCA runs laterally along sylvian fissure. The lenticulostriate branches arising from main stem of MCA supplies internal capsule and basal

ganglia. Finally MCA divides into inferior and superior branches. The inferior branch mainly supplies the lateral part of temporal lobe & inferior part of parietal lobes of brain. The superior parietal lobes & lateral aspect of frontal lobe are supplied by superior division of MCA.



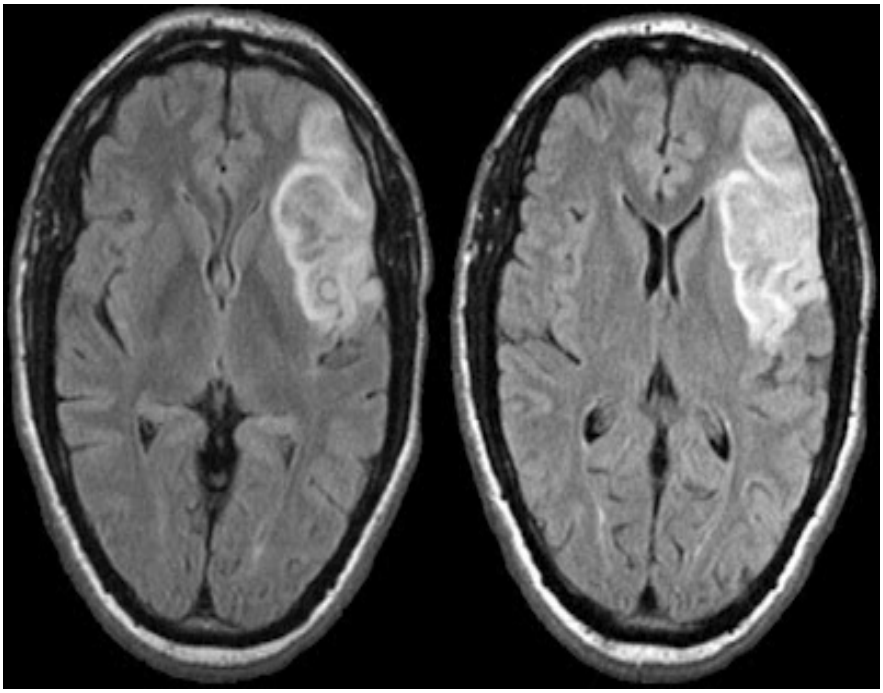
Middle cerebral artery & Segments

Clinical features of MCA infarct includes:

- C/L hemiparesis/hemiplegia
- C/L sensory disturbance
- I/L hemianopsia
- Gaze preference towards the same side of lesion

- Agnosia
- aphasia,(if infarct in the dominant hemisphere)
- hemineglect, (in some nondominant hemisphere lesions)

MCA Superior Division Infarction



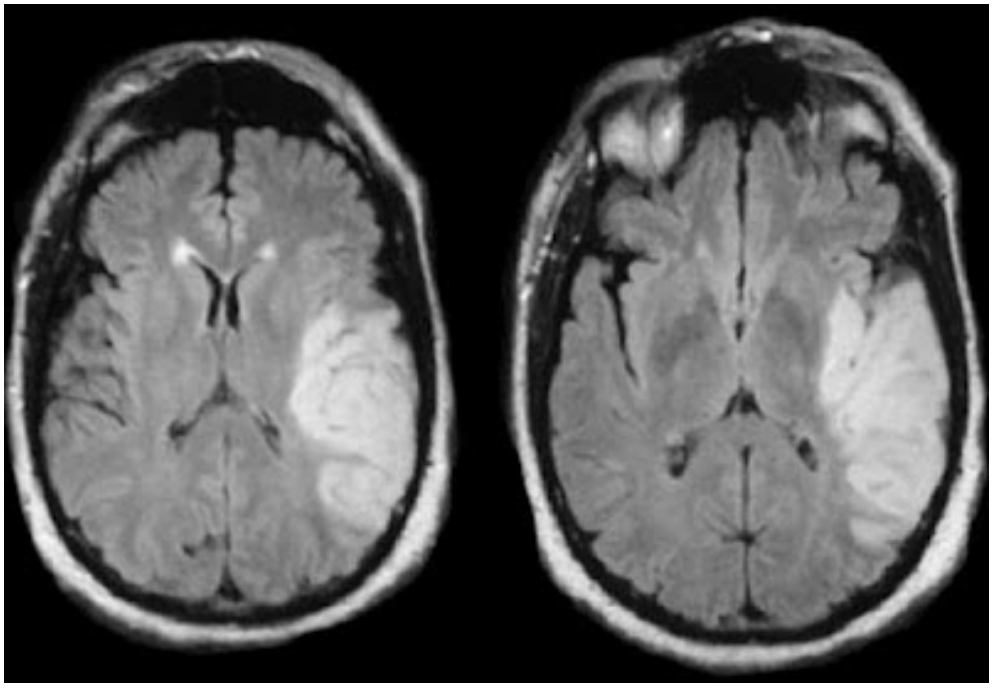
MRI (FLAIR) demonstrates infarction of left frontal lobe

(left MCA Superior Division territory)

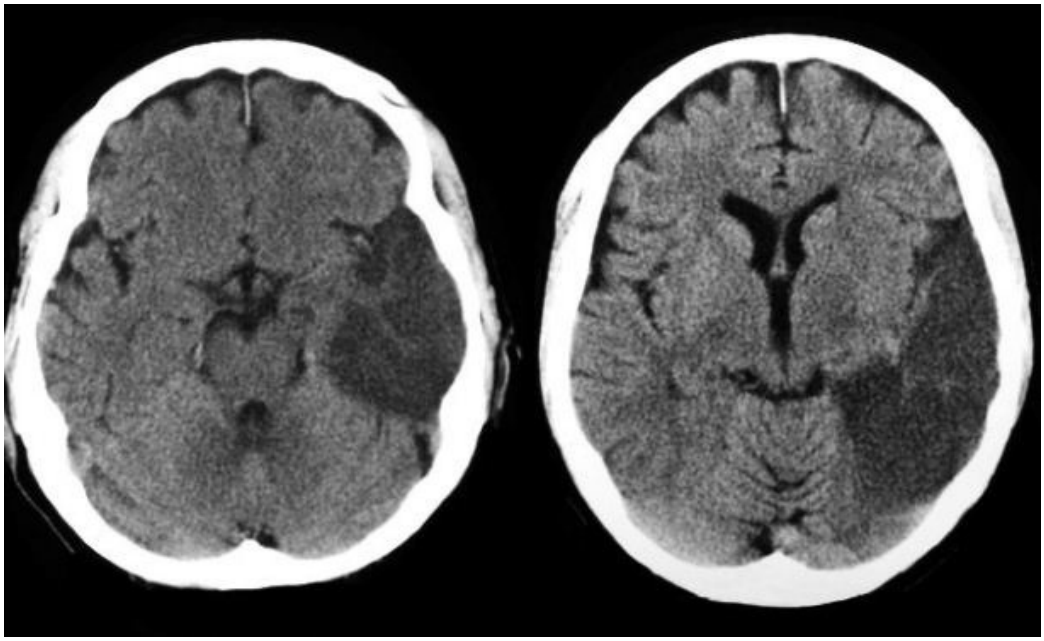
The superior division of the middle cerebral artery is one of the major sites of cardio embolic stroke. They typically cause contra lateral weakness of limbs (weakness more on upper limbs than lower limbs). similar pattern of cortical type of sensory disturbances; contra lateral visual field defects mainly involving lower field; Frontal eye field involvement results in gaze preference

to the ipsilateral side. Motor aphasia may be seen if dominant hemisphere involved. Non-dominant hemisphere lesion may produce neglect syndrome

MCA infarction (Inferior Division)



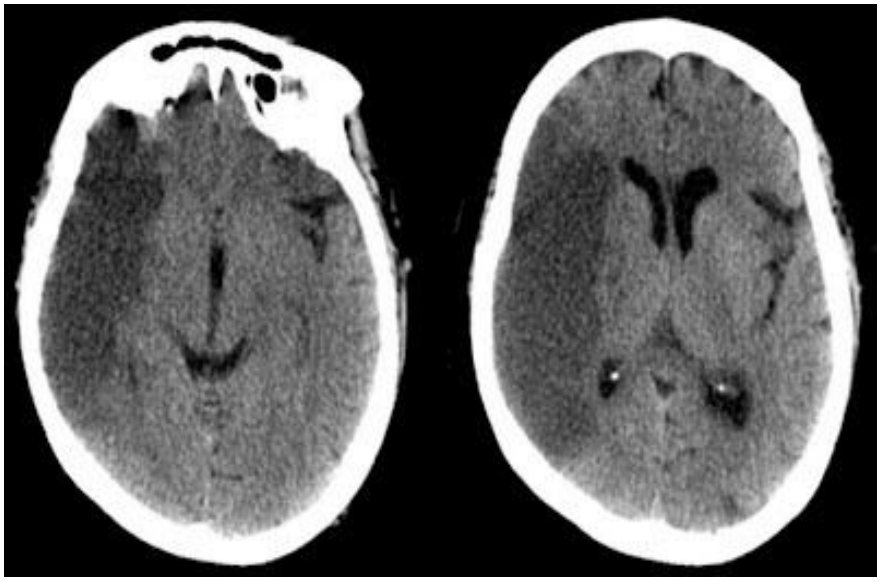
MRI brain (axial Flair): left temporal lobe infarction (left MCA Inferior Division territory)



CT brain: infarction in left temporal lobe (below the Sylvian fissure)- left MCA territory- Inferior Division

Inferior division MCA territory infarction: Sensory and motor system usually spared. They produce field deficits in contralateral eye mainly affecting upper field. ("pie in the sky" field deficit). Dominant hemisphere lesions causes sensory aphasia & non-dominant hemisphere lesions produces behavioural abnormalities and impaired visuospatial skills. Usually non dominant hemisphere lesions misdiagnosed initially with primary psychiatric disorder.

MCA infarction (Distal main stem)

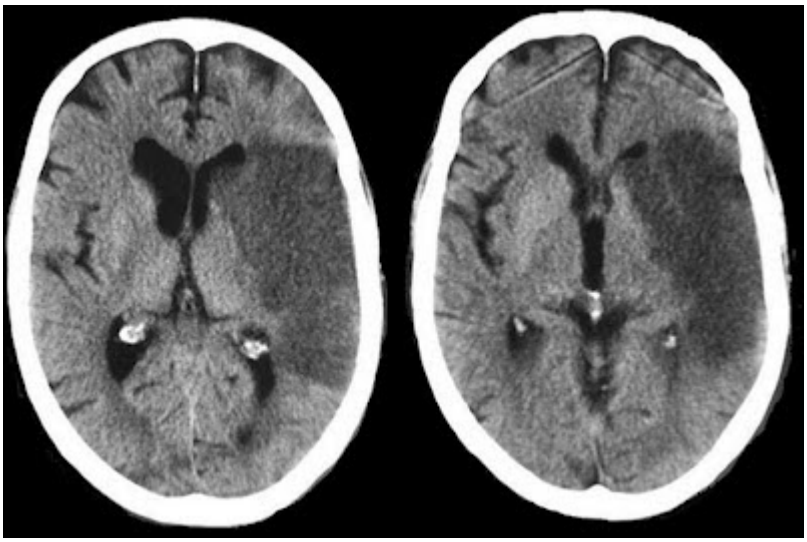


CT Brain : infarct involving Rt peri sylvian and adjacent insular cortex. Right basal ganglion not involved. (Rt MCA distal main stem territory)

Characteristic features – 1. Sparing of basal ganglia,

2. Distal MCA stem occlusion infarct result in contra lateral weakness (the lower half of face and upper extremities involved more than lower extremities), similar pattern of contra lateral hemi sensory loss or and visual field defects in opposite eye. Global aphasia may be seen if dominant hemisphere is involved. Non-dominant hemisphere lesions will present with neglect syndrome.

MCA Infarction (proximal stem)



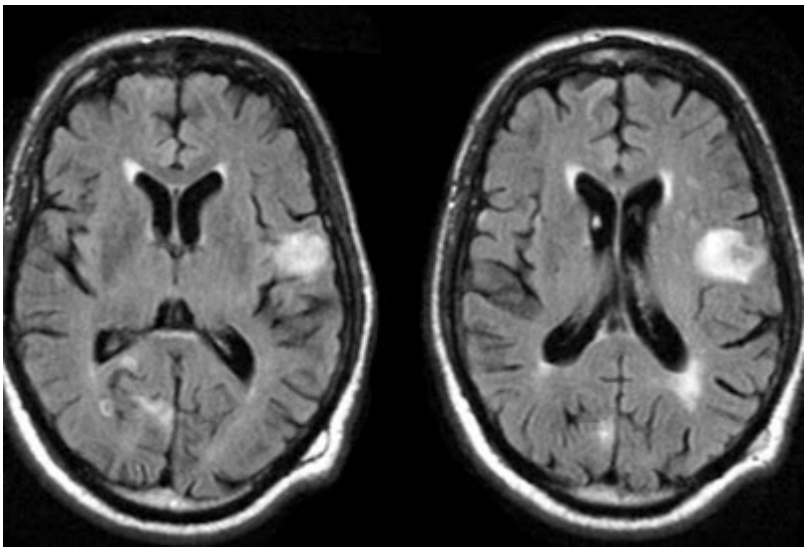
CT Brain shows an infarction t involving Lt perisylvian & insular cortex with involvement of basal ganglia (***left MCA territory -proximal mainstem***)

Characteristic features

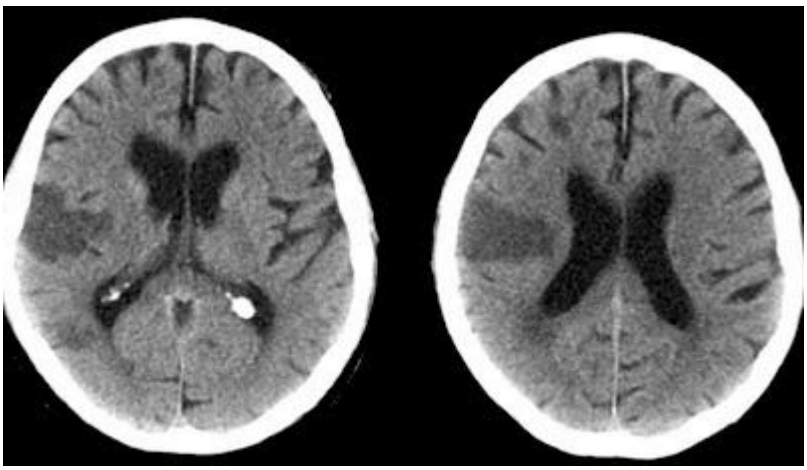
In proximal main stem lesion, there is weakness with equal involvement of upper and lower extremities. This is due to involvement of lenticulostriate arteries, causing internal capsule infarction.

In contrast to this, in distal main stem MCA infarction, internal capsule is spared.

MCA Infarction-cortical branch



MRI Brain (Axial FLAIR): infarction Lt precentral cortex –left MCA territory(cortical branch).



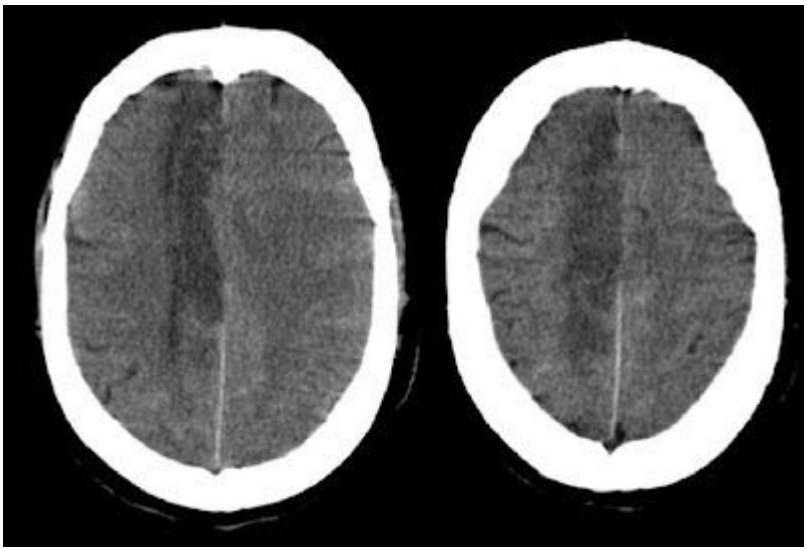
CT brain demonstrates infarction of Rt frontal pre central cortex - right MCA –cortical branch

ACA infarction:

Anterior cerebral artery (ACA) infarct usually affects functions of frontal lobe.

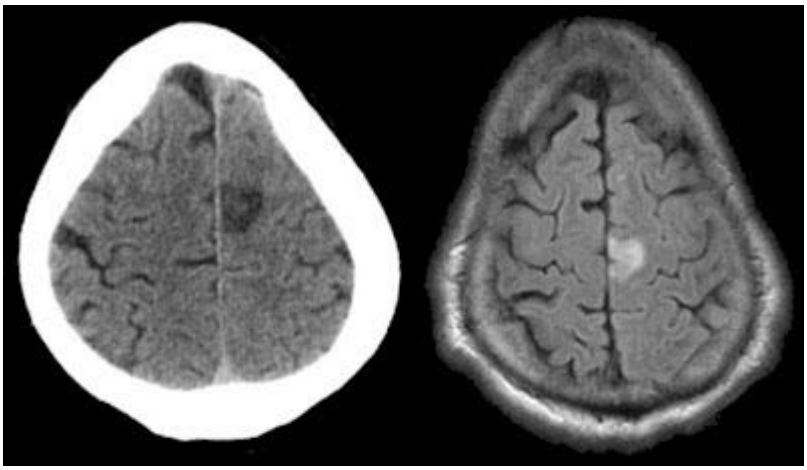
- Disinhibition, impaired judgement, inappropriate behaviour
- Presence of primitive reflexes like grasping, sucking reflexes
- C/L weakness (more in lower limbs when compared to upper limb)
- Impaired bladder functions

ACA Infarction- radio imaging



CT brain :infarction of para sagittal areas of Rt frontal lobe-

ACA territory (Rt)

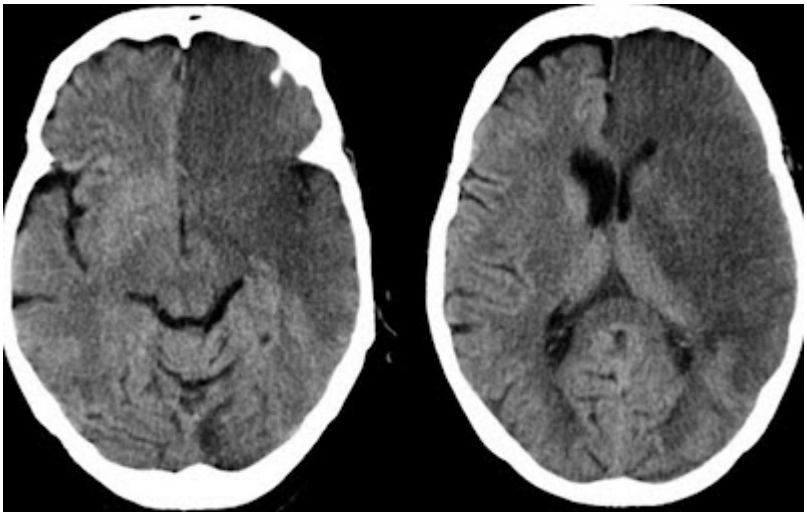


CT Brain &MRI (FLAIR): Small infarction in *Lt medial frontal lobe*

Characteristic features:

Infarction in medial frontal lobe predominantly causes weakness of the contralateral leg.

MCA - ACA Infarction



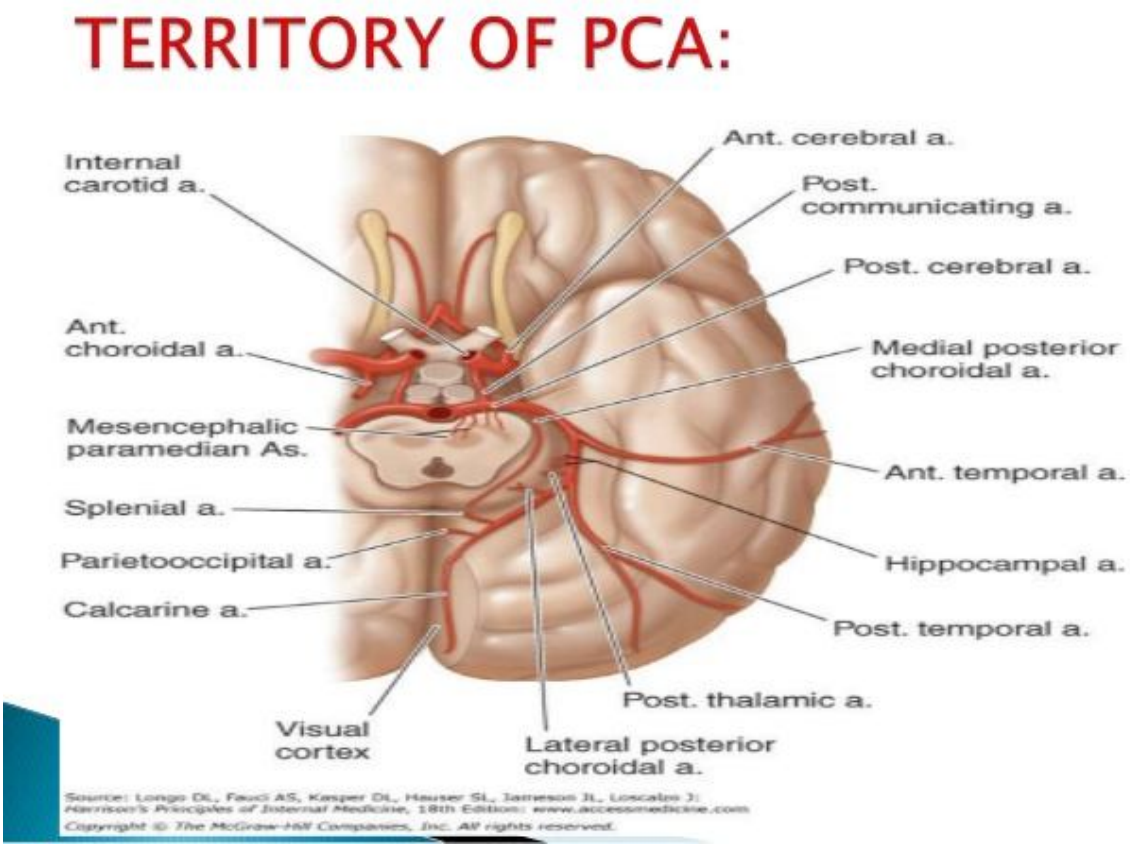
CT Brain: sub acute ischemic infarction - left MCA and ACA territory.

POSTERIOR CIRCULATION STROKE

Posterior circulation stroke has a wide variety of clinical symptoms and it is difficult to localize it. Clinical features include:

- Vertigo
- Nystagmus
- Double vision &field defects
- Dysphagia
- Dysarthria
- Facial hypesthesia
- Syncope
- Gait abnormalities

A classical finding in posterior circulation stroke is the presence of crossed findings- I/L cranial nerve deficits and C/L motor deficits.



The basilar artery divides into right and left posterior cerebral arteries. Posterior cerebral artery supplies inferomedial part of temporal lobe & medial part of occipital lobe. Cerebral peduncle & thalamus are supplied by perforators which arises from proximal part of PCA.

Infarctions in the PCA territory usually produces contralateral hemianopsia.

But other clinical features depend on the thalamoperforator involvement.

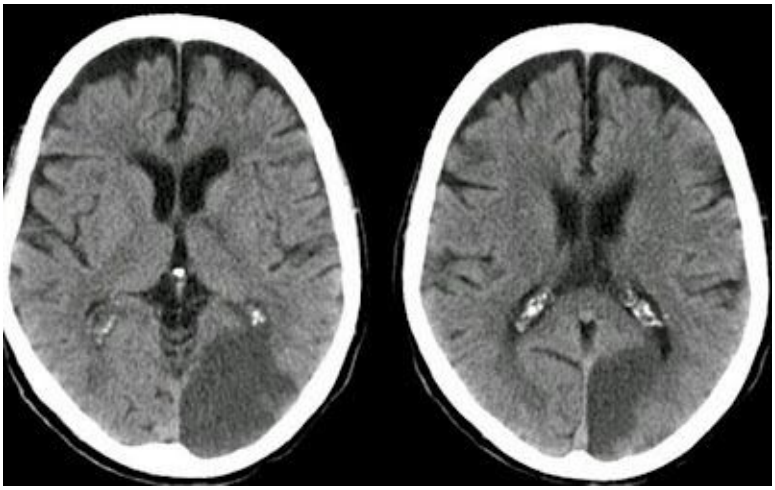
PCA infarction

Clinical features includes-

- C/L homonymous hemianopsia
- Cortical blindness

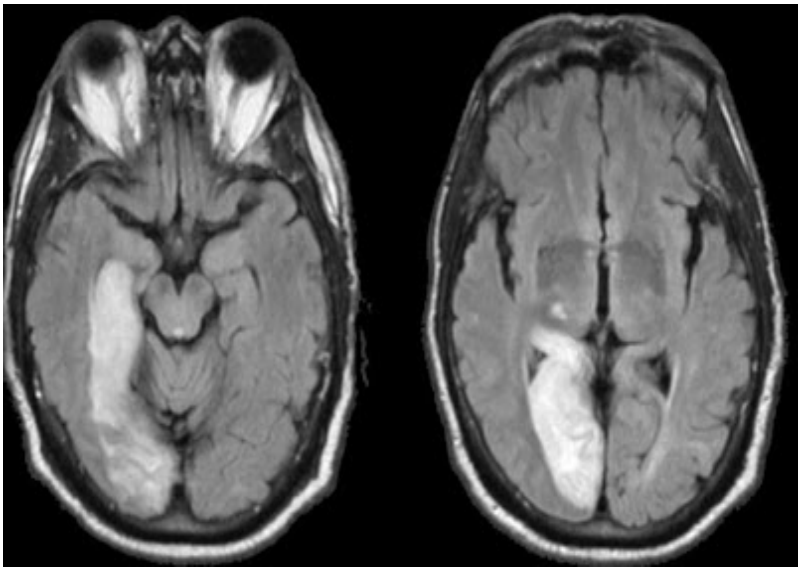
- Visual agnosia , Palinopsia, micropsia and macropsia
- acalculia, finger agnosia, agraphia and right/left confusion
- Paramedian thalamic infarction-memory impairment, lethargy to coma, behaviour abnormalities.

PCA Infarction-cortical branch



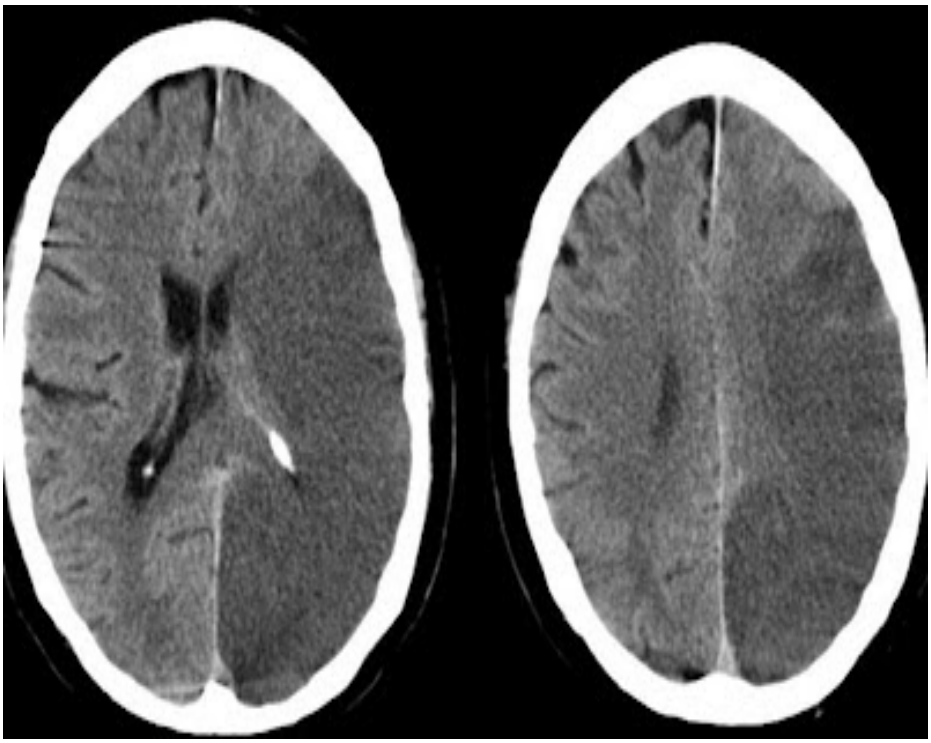
CT brain: left occipital infarction.

PCA Infarction-proximal



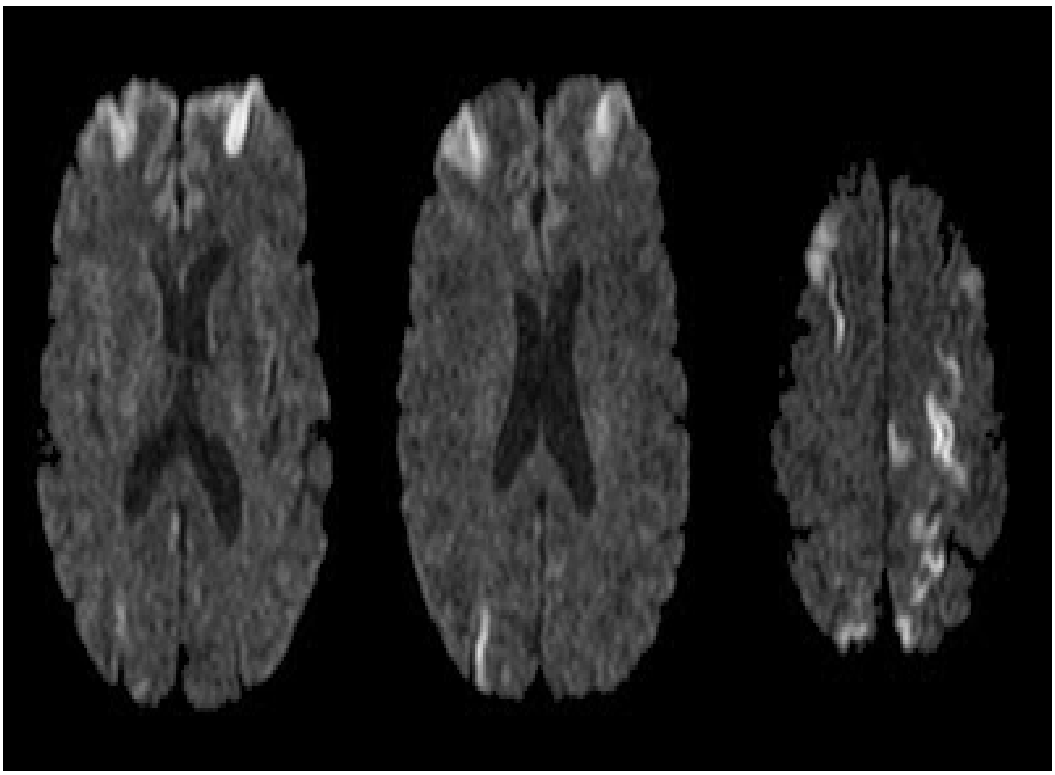
MRI (FLAIR)-infarction t of Rt thalamus, medial occipital and medial temporal lobe. (proximal PCA territory-Rt)

MCA - PCA Infarction



CT Brain -an infarction in the distribution of Lt MCA and PCA

Watershed Infarction



MRI Brain (diffusion: infarcts with restricted diffusion involving bilateral fronto parietal and parietal cortex. ACA - MCA watershed anteriorly and MCA - PCA watershed posteriorly.

Watershed infarcts between two vascular territories typically occur following hypotensive episode or shock. Usually they are bilateral. The stenotic lesions of proximal vessels may produce unilateral infarcts in the watershed zone.

The anterior watershed infarct between Anterior &Middle cerebral artery leads to a characteristic weakness of the shoulder &hip girdle muscles of both sides often referred to as "the man in the barrel" distribution of weakness (due to involvement of particular areas of the motor homunculus).

The posterior watershed infarction between middle & posterior cerebral artery result in bilateral vision abnormalities including-

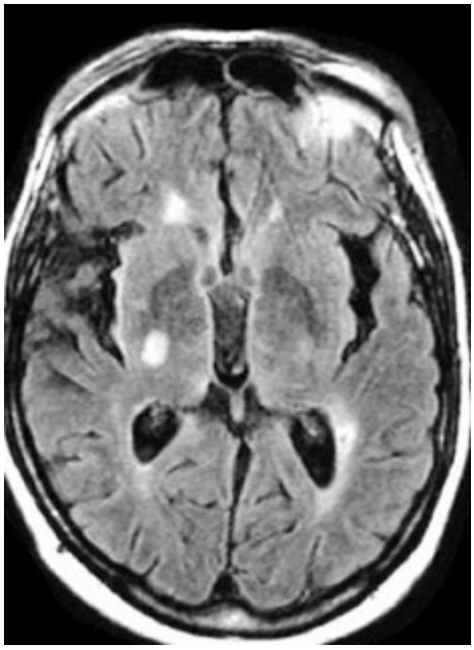
1. cortical blindness,
2. Anton's syndrome characterized by cortical blindness with denial/confabulation
3. Balint's syndrome - asimultagnosia, optic ataxia, and gaze apraxia.

CT brain: infarction of left basal ganglia along deep border zone

A deep watershed area lies between lenticulostriate branches and cortical branches of Middle cerebral artery. The lesion characterized by an infarct in basal ganglia, corona radiata or peri ventricular white matter.

Lacunar Infarction:

Lacunar infarct involves small, perforating branches in deep subcortical areas in brain parenchyma, usually lesions in internal capsule, basis pontis or thalamus. These are usually from 0.2-2cm in diameter. They are usually seen in hypertensives & diabetic patients. They may be classified as pure motor, pure sensory and ataxic hemiparetic types. Usually they will not affect cortical functions



MRI Brain: lacunar infarct in posterior limb of the internal capsule.

Diagnostic Considerations-conditions mimicking stroke

Before confirming the diagnosis of stroke we have to rule out other possible causes. The most frequent stroke mimics include the following:

- Convulsions (23% from ROSIER data)
- Sepsis (10%)
- Space occupying lesions
- Metabolic causes, such as hyponatremia and hypoglycemia
- Positional vertigo
- Conversion disorder
- Migraine

Transient ischemic attack

Transient ischemic attack (TIA) is defined as acute onset of reversible neurologic deficit, lasting for <24 hours, that results from focal cerebral, spinal cord, or retinal ischemia and is not associated with acute tissue infarction. Around 80% of TIAs resolve within one hour. Among TIA patients 10% will develop stroke within three months symptoms rather than true TIAs.

Work up

Brain imaging is essential to confirm the diagnosis of cerebrovascular accident. Non contrast computerized tomography is the initial investigation to confirm our clinical diagnosis. It helps in ruling out hemorrhagic lesions.

MRI with magnetic resonance angiography (MRA) used to study the structural details and early cerebral edema. **American Academy of Neurology** guidelines recommended DWI over non contrast CT scan for the diagnosis of acute ischemic stroke <12 hours of onset (level A).

Carotid duplex scan can be used.

Digital subtraction angiography is used as a definitive method for demonstrating vascular lesions.

Laboratory studies

A complete blood count (CBC) and random blood sugar, renal function test, urine routine examination.

The following investigations can be done if clinically indicated.

- Cardiac biomarkers
- Coagulation profile
- Protein C, Protein S, Antithrombin III assay, factor V leiden analysis
- Peripheral smear study
- C-ANCA, P-ANCA, complement assay

- Toxicology screening
- Fasting lipid profile
- ESR
- Antinuclear antibody (ANA)
- RA Factor
- Serum homocysteine level

OBJECTIVES OF THE STUDY

To study the clinical profile & frequency of association of various risk factors in acute ischemic stroke patients of Govt. Vellore medical college, Vellore.

MATERIALS AND METHODS

STUDY DESIGN –Hospital based cross sectional study

STUDY PERIOD: August 2014 – July 2015

STUDY POPULATION:

This study was conducted among 100 ischemic stroke patients who were admitted in medical ward Govt. Vellore medical college hospital, Vellore.

INCLUSION CRITERIA:

All patients of ischaemic stroke (on CT or MRI)

EXCLUSION CRITERIA:

Patients <18 yrs old and >80 yrs old.

Patient with other forms of stroke like ICH / SAH / post traumatic

Neoplasms (primary or secondary)

CNS infections

DATA COLLECTION:

After obtaining informed consent, detailed history, clinical examination, lab investigation reports were entered in the proforma specially designed for this study

LABORATORY INVESTIGATIONS:

RBS

Urine sugar

Lipid profile

ECG

Echocardiogram

CT brain/MRI brain

COLLABORATING DEPARTMENTS:

Department of radiology

Department of cardiology

Department of biochemistry

Ischemic Stroke was defined as focal neurological deficit due to vascular lesions that is due to cerebral infarction, confirmed on neuro imaging, resulting in partial or complete loss of motor and sensory activities. Patients meeting the criteria for stroke irrespective of sex were included. The presence or absence of following data was recorded for all the patients in a data extraction proforma-

1) Age and sex of the patient
2) Clinical features - a)weakness- right or left hemiparesis or hemiplegia,
   b)cranial nerve involvement
   c)speech involvement-dysarthria or aphasia
   d)altered sensorium
   e)other features-seizures, gait disturbances
3) Presence of Hypertension
4) Diabetes mellitus
5) Association with smoking
6) Presence of Dyslipidaemia
7) Presence of Obesity

8) Presence of heart diseases or atrial fibrillation

9) Alcoholism

10) Family income

11) Past history of TIA or stroke

12) Family history of stroke

Hypertension: As per JNC-7 criteria (stage I hypertension as systolic BP between 140-159 mmHg and diastolic BP between 90-99 mmHg, stage II hypertension as systolic BP >160 mmHg and diastolic BP 100 mmHg or more) patient diagnosed as hypertension and on treatment were considered as hypertensive. Patients who were not previously diagnosed as hypertension or not on any anti-hypertensive medications but presented with increased blood pressure at the time of presentation due to cushing's reflex were not defined as hypertensive.

Diabetes mellitus: According to American diabetes association, patient is to be considered diabetes when random blood sugar is above 200 or glycosylated haemoglobin is greater than 6.5 or fasting blood sugar more than 126 and post prandial blood sugar more than 200. So when patient satisfied above criteria or on any oral anti diabetic drug/ insulin regimen were considered as diabetes.

Smoking: A person who smoked 100 or more cigarettes during his lifetime was considered as a smoker. A "current smoker" was defined when he/ she smokes 1 cigarette/bedi per day for 3 months or more duration or consumes tobacco in different form.

"Never smoker," is a person who had not comes in to the criteria of current smoking person or ex-smoker.[75]

Dyslipidaemia: Patient is considered to be dyslipidaemic when he /she was previously diagnosed as dyslipidaemia or any anti-cholesterol drug or total cholesterol more than 200 during fasting and triglycerides more than 180, LDL more than 100 in current blood investigation.

Cardiovascular causes: Patient is said to have cardiovascular disease when he/she previous had coronary artery disease and its complication or undergone coronary artery bypass surgery or per cutaneous coronary intervention or symptoms suggestive of angina and his/her electrocardiogram showing changes suggestive of ischemia/infarction like presence of pathological Q wave /ST segment depression that meet criteria for ischemia or hypertensive patient with left ventricular hypertrophy proved either by echocardiogram/electrocardiogram showing increased QRS voltage and meeting criteria for LVH or echocardiogram showing dilated cardiac chambers or any cardiac condition causing atrial fibrillation.

Family History: Family is considered significant when his/her sibling/parents/first degree relative had suffered from stroke or transient ischemic attack previously and undergone treatment.

Obesity: Obesity is defined as patient with BMI > 30 kg/sqm.

Socioeconomic status: Based on BG Prasad's classification of socioeconomic status (updated 2014) patients were divided into 5 income classes. Per capita income calculated by dividing total family income with total

number of family members.

Class 1–per capita income (INR) >5410

Class 2-per capita income Rs 2700 -5410

Class 3-per capita income Rs 1620-2699

Class 4-per capita income Rs 810-1619

Class 5-per capita income upto Rs 809/month

TIA: TIA was defined as focal neurological deficit of sudden onset with complete recovery within 24 hrs.

Method Of Analysis

Patient's clinical presentation, vessel involvement, risk factors and their respective percentage was calculated. The data was compared with the studies carried out worldwide and in the Indian subcontinent and noted for any differences in the association of risk factors.

Ethical Issues

1. The objectives and procedure of the study was explained to all patients.
2. Informed consent was taken from all patients willing to participate in the study.
3. The option to opt out of the study was kept open without any clause.
4. Complete confidentiality regarding patient information was maintained through all stages of the study.

RESULTS

Between Aug 2014 and July 2015, 100 patients with ischemic stroke admitted at Government Vellore medical college hospital were studied. The male to female ratio was 3:1 (N.=100, Males-76: Females-24). Out of these patients only 22% (n=22) were ≤45 years and 78% (n=78) were >45 years. The maximum frequency of stroke was found for ages more than 45 years.

Table 1- Age wise distribution of ischemic stroke patients

Age group	**Sex**		**Total**
	Male	**Female**	
18-30 years	5	2	7
31-45 years	12	3	15
>46 years	59	19	78
Total	76	24	100

Figure 1: Age wise distribution of ischemic stroke patients

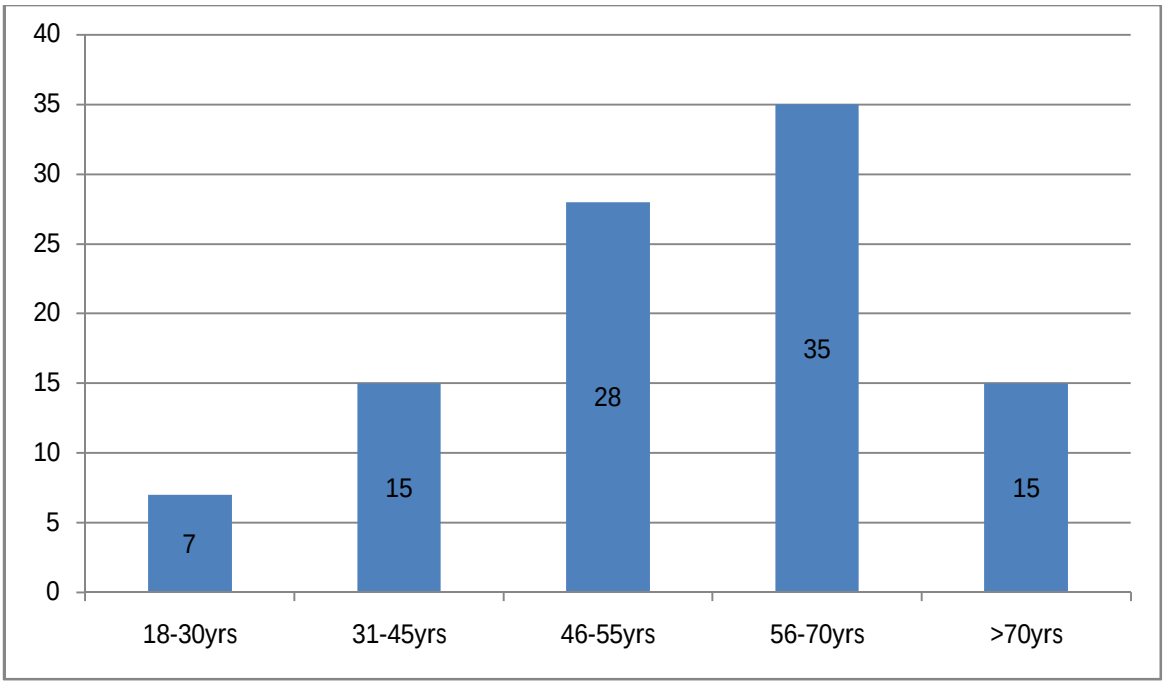


Table 2: Distribution of ischemic stroke patients by socioeconomic status

Socio economic class	Class 5	Class 4&3	Class 2
No. of Patients	66	33	1
%	66%	33%	1%

Maximum number of patients (66%) were in the low income group (class 5). 33% of patients were in middle group (class 4 &3) and one patient was in class 2 (B G Prasad classification) (Table 2).

Figure 2: Socioeconomic status of stroke patients(B G Prasad classification 2014)

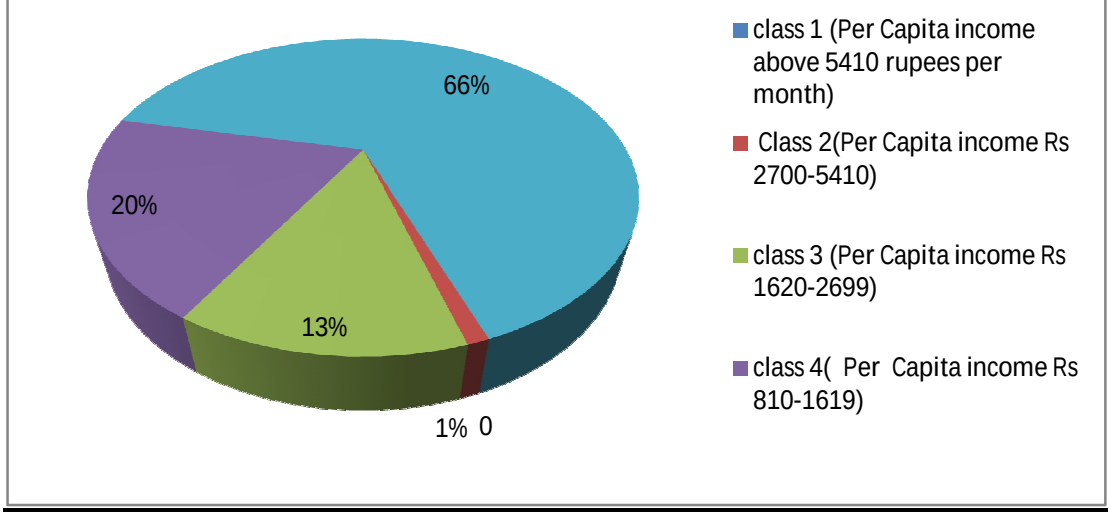


Table 3: Frequency of clinical features in ischemic stroke patents

Clinical Features	**Percentage**
Motor weakness (Hemiplegia/Hemiparesis/Monoparesis)	88% (Rt sided 42, Lt sided 46)
Speech involvement (Aphasia or Dysarthria)	44% (38% Dysarthria, 6% Aphasia)
Cranial nerve involvement	67% (63%UMN 7th nerve, 2% LMN 3rd nerve, 1%LMN 7th & 12th nerve with gaze palsy and 1% LMN 9 &10 Nerves
Altered sensorium	16%
Seizures	7%
Gait disturbances/Cerebellar signs	3%

Figure 3: Frequency of clinical manifestations in stroke patients

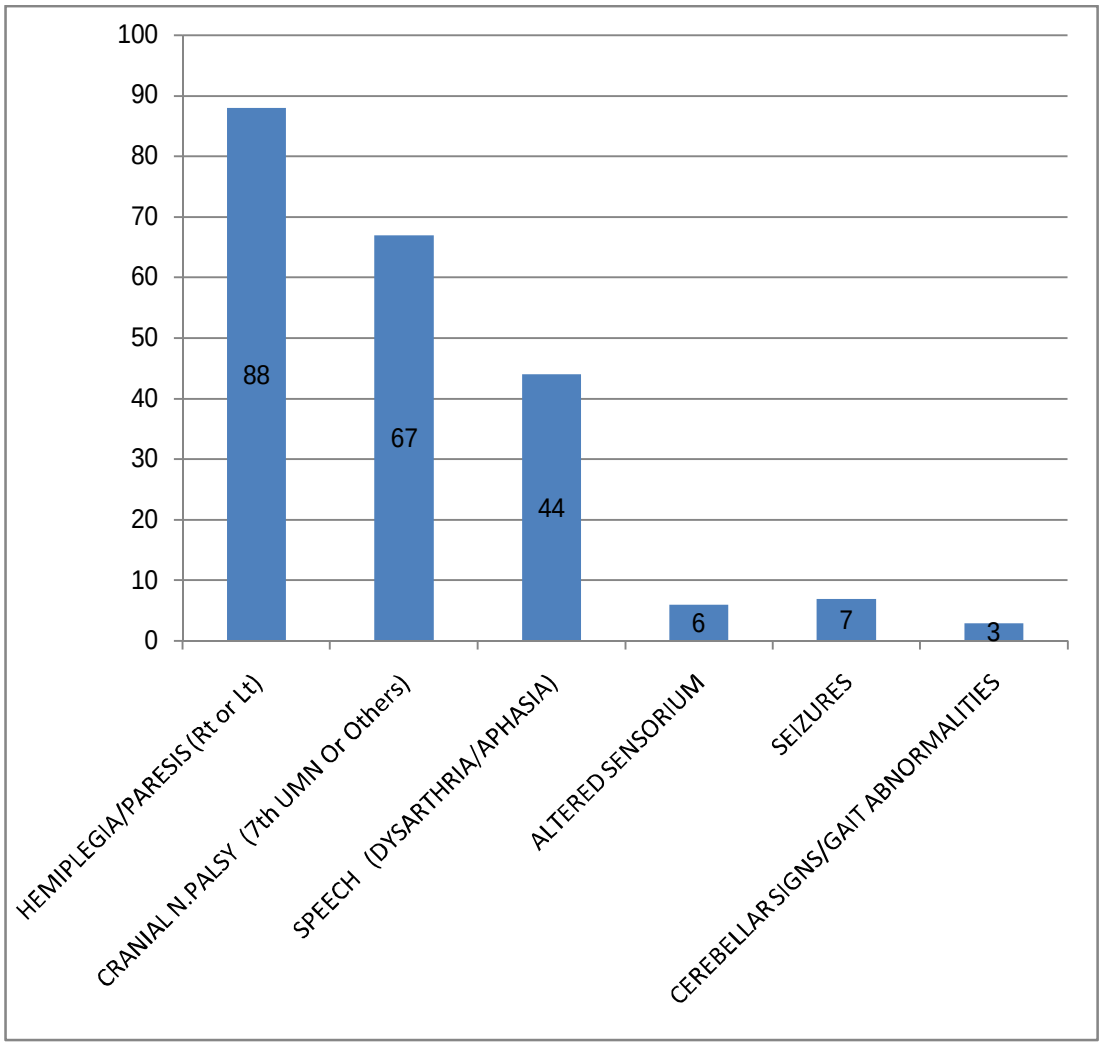
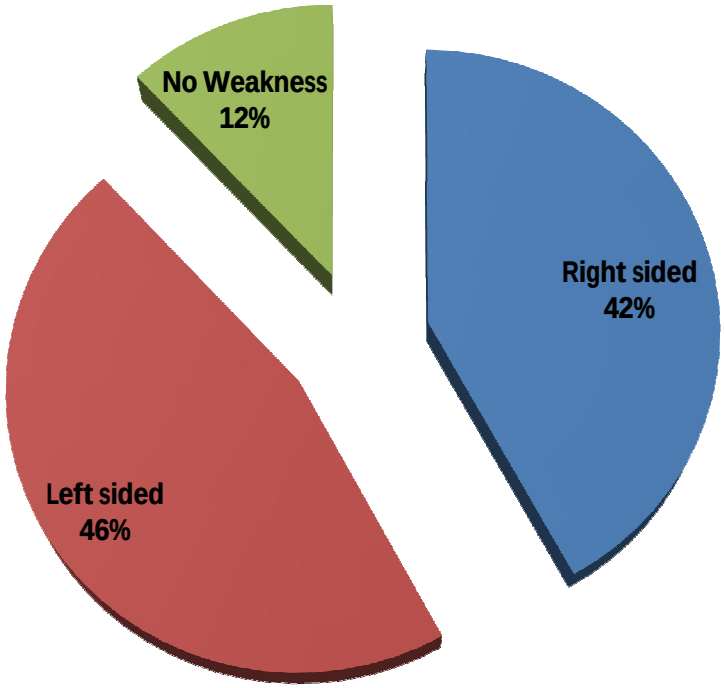
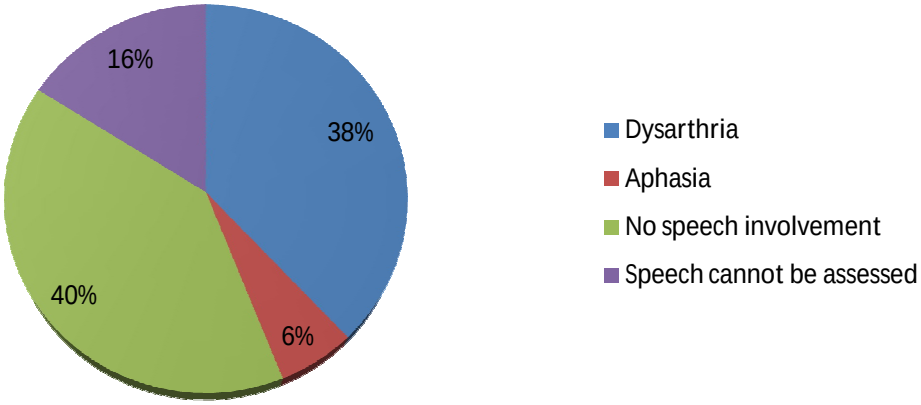


Figure 4: Frequency of Hemiplegia/Hemiparesis in ischemic stroke patients



- In our study, most common clinical feature was hemiplegia/hemiparesis.
- Eighty eight percent (88%) patients had hemiplegia/hemiparesis.
- Among these left sided weakness was more common (46% patients had left sided hemiplegia/hemiparesis)
- 12% patients had stroke without any evidence of motor weakness.

Figure 5: Speech Involvement in ischemic Stroke patients

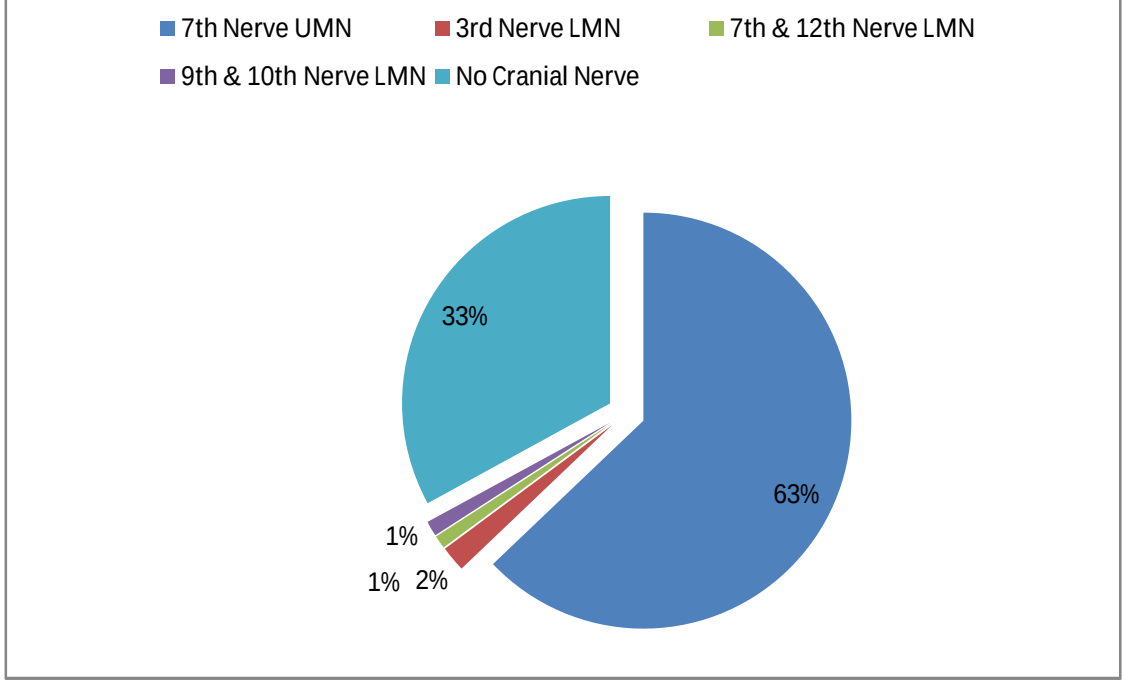


Speech disturbances found in 44% patients, 38% patients had dysarthria & 4% patients had motor aphasia.

Another 2% of patients affected with global aphasia.

In 100 patients 40% had normal speech (speech couldn't be assessed in 16% of patients because of altered sensorium)

Figure 6: Cranial nerve involvement in Stroke patients



The second most common clinical feature was cranial nerve involvement (67%). UMN fibres of facial nerve involvement was identified in 63 patients (63%). One patient had 9^{th} &10^{th} (LMN) cranial nerve involvement. Another one patient had 7^{th} &12^{th} cranial nerve involvement (LMN).Third nerve is involved in two patients.

Other clinical features:

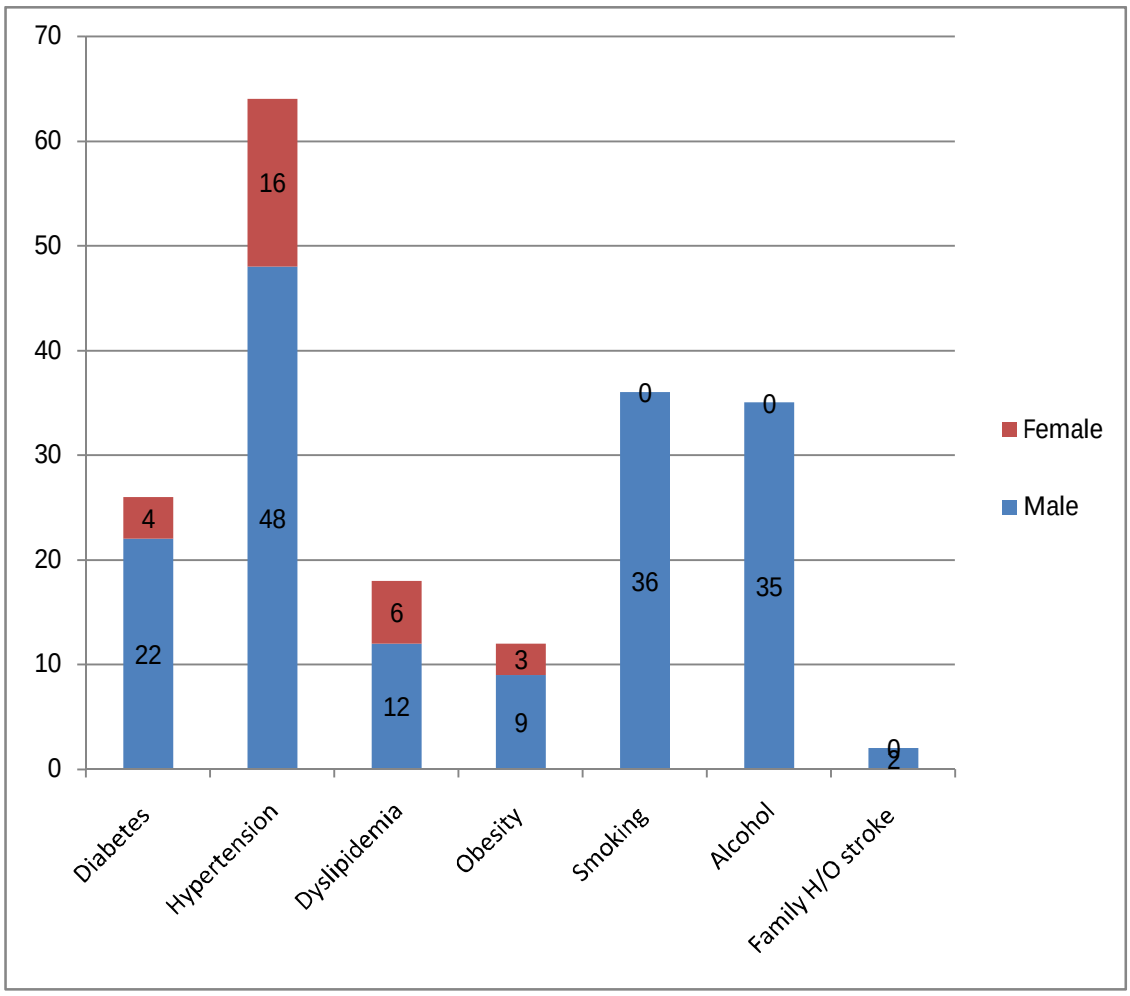
- Among 100 stroke patients altered sensorium was found in 16 patients
- 7% of patients had seizures
- Three patient (3%) presented with cerebellar signs

Table 4: frequency of risk factors in ischemic stroke patients

Risk Factors	**No. of patients**	**%**
Hypertension	64	64
Diabetes	26	26
Smoking	36	36
Dyslipidemia	18	18
Alcohol	35	35
Family history of stroke	2	2
H/O Coronary heart disease/RHD/cardiomyopathy/AF	15	15
Past h/o TIA or Stroke	10	10
Obesity	12	12

Risk factors include systemic hypertension, chronic smoking, chronic alcohol intake, Diabetes Mellitus, significant cardiovascular disease, significant positive family history, dyslipidaemia, past transient ischemic attack , past history of stroke and obesity.

Figure 7: Risk factors of ischemic stroke patients according to sex



The commonest modifiable risk factor in our study was hypertension in 64% patients.

Smoking was second common modifiable risk factor in our patients (36%). Alcohol consumption was the third common modifiable risk factor (35 %).

Smoking &Alcoholism were identified only in male population. The data was not sufficient to quantify the amount and duration of alcohol consumption. However, considering the alcohol habit of population in our

patients it should be considerably high.

Diabetes mellitus (DM) which is an established risk factor was found in 26% patients. Among all the stroke patients 16% were known diabetic and hypertensive.

A total of 15% patients had concomitant heart disease (coronary artery disease/RHD/Cardiomyopathy/AF).

Eighteen percent patients had dyslipidaemia. Family history of stroke or TIA was present in 2% of patients.

12% of patients were obese and 10% of patients had previous history of stroke or TIA.

Table 5: Age wise distribution of risk factors in ischemic stroke patients

RISK FACTORS	≤30 YEARS n=7, N(%)	31-45 YEARS n=15, N(%)	≥46 YEARS n=78, N(%)	p VALUE
SHT **#	0(0)	3(20)	61(78.2)	0.001
DIABETES#	1(14.3)	2(13.3)	23(29.5)	0.326
SMOKING** #	0(0)	1(6.7)	35(44.9)	0.002
ALCOHOL*#	0(0)	3(20)	32(41)	0.039
DYSLIPIDEMIA*#	0(0)	0(0)	18(23.1)	0.045
OBESITY#	0(0)	0(0)	12(15.4)	0.146

Chi square test used to compare the groups *p<0.05, ** p value <0.005, # Fischer's exact test was used.

There is a significant difference (p=0.001) in association of systemic hypertension and incidence of ischemic stroke with respect to various age groups. In patients with age ≥46 years, systemic hypertension plays a major role in the incidence of stroke. Smoking, Alcoholism, Dyslipidemia also has a significant difference in association with incidence of ischemic stroke with respect to different age groups.

As age advances systemic hypertension, smoking, alcoholism, dyslipidemia plays a major role in the incidence of ischemic stroke. There are no such association between stroke, diabetes & obesity with respect to age. (Table 5)

Table 6: Risk factors of ischemic stroke according to sex of the patients

RISK FACTORS	**MALE n=76, N(%)**	**FEMALE n=24, N(%)**	**P VALUE**
SHT[#]	48(63.2)	16(66.7)	0.755
DIABETES[#]	22(28.9)	4(16.7)	0.232
SMOKING[** #]	36(47.4)	0(0)	0.001
ALCOHOL[**#]	35(46.1)	0(0)	0.001
DYSLIPIDEMIA[#]	12(15.8)	6(25)	0.306
OBESITY[#]	9(11.8)	3(12.5)	0.931

Chi square test used to compare the groups. *p<0.05, ** p value <0.005, [#] Fischer's exact test was used

There are no significant difference in the association of Hypertension Diabetes, Dyslipidemia & obesity in the incidence of ischemic stroke with respect to sex. Smoking &Alcoholism found only in male cases. Significant association was found between the incidence of stroke with alcohol and smoking (p=0.001). The higher incidence of stroke in male cases may be due to smoking & alcoholism which were identified only in males.

Table 7: Vascular territory involvement in stroke patients

S.No.	Arteries involved	%
1	MCA alone	92%
2	ACA alone	2%
3	PCA alone	4%
4	MCA with PCA	1%
5	MCA with ACA	1%

The middle cerebral artery was most common arterial territory involved in our study. Around 92% of patients had middle cerebral artery infarction. Posterior & Anterior cerebral artery was involved in 4% &2% of patients respectively .One patient had MCA with ACA territory infarct, while another one patient had MCA with PCA infarction.

DISCUSSION

Stroke is major public health problem which has a significant morbidities and mortalities. Worldwide, it is the third most common cause of death in adults. Stroke occurs predominantly in males at late years of life. Several studies documented that systemic hypertension, diabetes mellitus, hyperlipidemia, ischemic heart disease, atrial fibrillation, smoking and long standing alcohol intake are contributing factors for stroke. The prevalence of risk factors varies in different population. Despite numerous prior studies of stroke, risk factors much remains unknown and several inconsistencies continue to exist. However the minor differences in the prevalence of stroke risk factors in different communities are probably due to differences in culture, disease patterns, living habits and distribution of various ethnic groups.

Various modifiable and non modifiable risk factors were studied and analysed in this study.

Age and Ischemic Stroke Incidence:

In this study youngest patient was 21 years and oldest was 79 years old. Elderly people are the most vulnerable group for developing stroke. The stroke incidence is high in the age group of 56-70 years of age. It is seen that 78% of the sufferers were in the age group ≥46 years and incidence increased with increasing age as depicted in the Bar graph. **The mean age in our study is 55.86±14.98 years.** It closely resembled **Aiyar et al[76]** study which showed mean age of affected person was 55.39 years. Young stroke (age ≤45 years) comprised of 22% of all ischemic stroke patients. Our study closely correlates

with study done by **Gauri et al** (19%),[78] **P. Chitrambalam et al (20%).**[77]

No	Studies on Stroke	Mean Age
1.	Naik M,Rauniyar R.K.,Sharma U.K.,et al [79]	58.27 years
2.	Aiyar et al [76]	55.39 years
3.	Pinhero et al [80]	54.85 years
4	R P Eapen et al [81]	57 years
5	Present study	55.86 years

Male predominance:

In the above study males were most commonly affected by stroke which is supported by the studies conducted in western countries.[82-84] It also correlates with study done by **Aiyar et al [76], Pinhero et al** [80] and **R P Eapen et al** [81] who found the incidence of stroke is more common in males than females.

S.no.	Studies	Male to female ratio
1	Aiyar et al [76]	1.9:1
2	R P Eapen et al [81]	2:1
3	Kay Sin Tan et al [85]	1.9:1
4.	Present study	3:1

However the ratio of 3:1 seen in this study which is higher than the studies conducted elsewhere. It could be due to lack of proper care to women and late admission in hospitals after it became severe illness. In our society, smoking and alcoholism in male population is high, which is not so common

among female population. In our study no cases of female with alcoholism or smoking had been reported. The **influence of smoking and alcoholism on stroke incidence has to be studied in detail.**

Socioeconomic status Stroke patient's economic status also analysed. It revealed that 66% stroke patients were belong to low income group (class 5 Prasad classification) which were considered per capita income less than INR 810 per month. In above study people from low income group mostly affected because upper class people mostly not admitted in government hospital which contradicted the results of **Chapman et al**[86] study**.** So the real incidence of stroke in relation to economic class varies in this study.

Modifiable Risk Factors:

In above study 64% of stroke patient had hypertension and which is found to be single most risk factor associated with stroke. This result correlating with **Benerjee TK et al**[83] study which was conducted in Calcutta on urban population in which systemic hypertension emerged as single most important risk factor.

No	Stroke studies	Systemic hypertension
1	Naik M,Rauniyar R.K.,Sharma U.K.,et al [79]	40.66%
3	R P Eapen et al [81]	32%
4	Kay Sin Tan, Jose C Navarro et al[85]	42.6%
5	Present study	64%

Diabetes mellitus is one well known, studied risk factor causing macrovascular complications. When compared with non diabetic patient stroke risk doubles in diabetes.[35] The above study reveals 26% stroke patient has diabetes. In Framingham study 10 to 14% person with stroke had diabetes. The higher prevalence seen in our study may be due to higher prevalence of diabetes in southern India from where most of the population under study hails. The data is in agreement with several other Indian studies.[86] 16% of patients had both Hypertension and Diabetes. From various studies it was concluded that strict control of blood pressure in diabetic patients will definitely reduces the incidence of stroke.[34]

Smoking appears as an important risk factor for ischemic stroke in this study. Thirty six patients (36%) were smoker among 100 stroke patients studied. The above study is correlating with **Donnan et al,[88]** where smoking was strongest risk factor causing ischemic stroke. In another study **Kaul et al[89]** observed 28% of stroke patients were smokers.

In our study, dyslipidemia noted in 18% of patients. It correlates with prior study done by **Eapen et al (17%).[81]**

Thirty five (35%) patients were found to be alcoholic. Our study correlates with study by **Naik M, Rauniyar R.K.,Sharma U.K.et al[79]** who found H/O alcohol intake in 30.5% of stroke patients. For cerebral infarction chronic heavy drinking and acute intoxication have been associated with an increased risk among young adults.[90] In older adults risk is increased among heavy-drinking men. Some studies have supported a J-shaped dose-response

curve between alcohol intake and ischemic stroke risk, with protection for those drinking up to 2 drinks per day and an increased risk for those drinking >5 drinks per day compared with non drinkers.[91] The deleterious effects of alcohol for stroke may occur through various mechanisms, including increasing hypertension, hyper coagulable states, and cardiac arrhythmias and reducing cerebral blood flow. The limitation of the study was that the daily quantity and the type of alcohol could not be specified.

In the present study, association between various risk factors and incidence of stroke with respect to age and sex were studied. When the age advances, there is a significant association between the stroke incidence and risk factors like hypertension, smoking, alcoholism and dyslipidemia. Except smoking and alcoholism, there is no significant difference in the association between stroke with respect to sex.

H/o past transient ischemic attack or stroke accounted for 10% of patient.

No.	**Studies**	**%**
1	Ukoha Ob et al[92]	16.2%
2	Abdu-Alrhaman Sallam et al[93]	12.2%
3	Present study	10%

Heart diseases including coronary artery disease, cardiomyopathy, valvular heart diseases were observed in 15% of cases.

In our study hemiplegia was the most common clinical feature. It was reported

in 88% of patients, followed by cranial nerve involvement in 67% patients. Speech disturbance was noted in 44% patients. These features correlate with most of the studies.

Regarding vascular territory 92% patients had isolated middle cerebral artery infarcts. Posterior cerebral artery is involved in 4% cases. Only 2% patients had anterior cerebral artery involvement. One patient presented with middle & anterior cerebral artery stroke. Another one had middle & posterior cerebral artery involvement. Our study correlates with **Devichand and caroli et al** [94] who also found the middle cerebral artery involvement in 98% of patients.

CONCLUSION AND RECOMMENDATIONS

Between Sep 2014 and July 2015, 100 patients with ischemic stroke admitted at Government Vellore medical college hospital were studied

- The stroke incidence is high in the age group of 56-70 years of age. It is seen that 78% of the sufferers were in the age group >45 years
- Stroke is more common in males than females (ratio 3:1).
- Most of the stroke patients (66%) in our study belong to low income group.
- Most common clinical feature was hemiplegia/hemiparesis. Eighty eight percent (88%) patients had hemiplegia/hemiparesis. 12% patients had stroke without any evidence of motor weakness. Cranial nerve involvement noted in 67% patients and Speech disturbances found in 44% patients.
- Most common vascular territory involved is middle cerebral artery. Around 92% of patients had middle cerebral artery infarction.
- **In our study hypertension, smoking, alcoholism, dyslipidemia were significantly associated with stroke in patients with age group more than 45 years.**
- **In 100 total stroke patients 64% had hypertension and it is found to be high when compared to previous studies**. So screening programs have to be strengthened in our outpatient department to identify people

with undiagnosed hypertension and appropriate treatment should be initiated. Patient compliance to treatment should be ensured.

- With the help of non communicable disease control program hypertension and dyslipidemia should be identified. If these patients had additional risk factors like smoking and alcoholism they should be educated for stroke prevention. Along with diet modification and drugs patients should be motivated to discontinue smoking and alcoholism. It may reduce the incidence of future ischemic stroke in our population.
- Among 100 ischemic stroke patients 16% of patients had both Hypertension and Diabetes. Hypertensive patients who have diabetes mellitus should be followed up at more frequent interval and tight control of blood pressure in diabetic patients may reduce stroke incidence.
- When compare to other studies stroke incidence is very high in male population. Smoking and alcoholism identified only in male population. In total stroke patients, 36% of patients were smokers & 35% were found to be alcoholic. The role of smoking and alcoholism in the incidence of ischemic stroke has to be studied in detail.
- 10% of patients had past episode of TIA or previous stroke attack. So all patients with TIA should be followed up regularly and additional risk factors in these patients should be modified. Stroke patients should be advised for regular follow up. Risk of recurrence should be explained and secondary prevention measures should be intensified.

- 15% of patients had heart disease or atrial fibrillation. Patients who had previous myocardial infarction, cardiomyopathy or patients with valvular heart disease /AF should be monitored regularly. With regular follow up we can prevent stroke.

BIBLIOGRAPHY

1. Stroke in developing countries: epidemiology, impact, policy implications. University of east Anglia: School of international development; 2009 Aug. 32p.
2. Nagaraja D, Gururaj G, Girish N, Panda S, Roy AK, Sharma GRK et al. Feasibility study of stroke surveillance: Data from Bangalore, India. Indian J Med Res. 2009 Oct; 130: 396-403.
3. Feigin V, Lawes C, Bennet D, Barker-Cello S, Parag V. Worldwide stroke incidence and early case fatality in 56 population based studies: a systematic review. Lancet neurology. 2009; 8(4): 355-69.
4. Disease and injury regional estimates for 2004. Geneva, Switzerland: World Health Organization; 2005.
5. Preventing Chronic Diseases: A vital iinvestment. Geneva, Switzerland: World Health Organization; 2005.
6. World Health Report - 2007, from the World Health Organization; International Cardiovascular Disease Statistics (2007 Update), a publication from the American Heart Association.
7. Goldstein M, Barnett HJM, Orgogozo JM, Sartorius N, Symon L, Vereshchagin NV. Stroke—1989: Recommendations on stroke prevention, diagnosis, and therapy. Report of the WHO Task Force on Stroke and other Cerebrovascular Disorders. Stroke, 1989; 20(10): 1407–31.
8. Blank-Reid C. How to have a stroke at an early age: The effects of

crack, cocaine and other illicit drugs. Journal of Neuroscience Nursing. 1996; 28(1): 19–27.

9. APASS Investigators. Antiphospholipid antibodies and subsequent thrombo-occlusive events in patients with ischemic stroke. Journal of the American Medical Association. 2004; 291(5): 576–84.

10. Bader MK, Littlejohns LR. Editors. AANN core curriculum for neuroscience nursing. 4th ed. St. Louis: Elsevier; 2004.

11. Muir KW, Buchan AM, von Kummer R, Rother J, Baron JC. Imaging of acute stroke. Lancet Neurology. 2006; 5: 744–68.

12. Dhamija RK, Dhamiga SB. Prevalence of stroke in rural community—an overview of Indian experience. J Assoc Physicians India. 1998; 46(4): 351-4.

13. Brown RD, Whisnant JP, Sicks JD. Stroke incidence, prevalence, and survival: secular trends in Rochester, Minnesota, through 1989. Stroke. 1996; 27: 373–80.

14. Sacco RL, Boden-Albala B, Gan R. Stroke incidence among white, black, and Hispanic residents of an urban community: the Northern Manhattan Stroke Study. Am J Epidemiol. 1998; 147: 259–68.

15. Bousser M-G. Stroke in women: the 1997 Paul Dudley White International Lecture. Circulation. 1999; 99: 463–7.

16. Kittner SJ, Stern BJ, Feeser BR, Hebel JR, Nagey DA, Bucholz DW et al. Pregnancy and the risk of stroke. N Engl J Med. 1996; 335: 768–74.

17. Welin L, Svardsudd K, Wilhelmsen L, Lasson B, Tibblin G. Analysis of risk factors for stroke in a cohort of men born in 1913. N Engl J Med. 1987; 317: 521–6.

18. Liao D, Myers R, Hunt S, Shahar E, Paton C, Burke G et al. Familial history of stroke and stroke risk: the Family Heart Study. Stroke. 1997; 28: 1908–12.

19. Hrubec Z, Robinette CD. The study of human twins in medical research. N Engl J Med. 1984; 310: 435–41.

20. Brass LM, Isaacsohn JL, Merikangas KR, Robinette CD. A study of twins and stroke. Stroke. 1992; 23: 221–3.

21. Wolf PA. Cerebrovascular risk. In: Izzo JL Jr, Black HR, Goodfriend TL, et al. Hypertension Primer: The Essentials of High Blood Pressure. Baltimore, Md. Lippincott Williams & Wilkins; 1999.

22. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J et al. Blood pressure, stroke, and coronary heart disease, part 1: prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. Lancet. 1990; 335: 765–74.

23. Burt VL, Whelton P, Roccella EJ, Brown C, Cutler JA, Higgins M et al. Prevalence of hypertension in the US adult population: results from the Third National Health and Nutrition Examination Survey, 1988–1991. Hypertension. 1995; 25: 305–13.

24. Joint National Committee. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. Arch Intern Med. 1997; 157: 2413–46.

25. Veterans Administration Cooperative Study Group on Antihypertensive Agents. Effects of treatment on morbidity in hypertension, II: results in patients with diastolic blood pressure averaging 90 through 114 mm Hg. JAMA. 1970; 213: 1143–52.

26. Klungel OH, Stricker BH, Paes AH, Seidell JC, Bakker A, Voko Z et al. Excess stroke among hypertensive men and women attributable to under treatment of hypertension. Stroke. 1999; 30: 1312–8.

27. Kool MJ, Hoeks AP, Struijker Boudier HA, Reneman RS, Van Bortel LM. Short- and long-term effects of smoking on arterial wall properties in habitual smokers. J Am Coll Cardiol. 1993; 22: 1881–6.

28. Cruickshank JM, Neil-Dwyer G, Dorrance DE, Hayes Y, Patel S. Acute effects of smoking on blood pressure and cerebral blood flow. J Hum Hypertens. 1989; 3: 443-9.

29. Shinton R, Beevers G. Meta-analysis of relation between cigarette smoking and stroke. BMJ. 1989; 298: 789–94.

30. Wolf PA, D'Agostino RB, Belanger AJ, Kannel WB. Probability of stroke: a risk profile from the Framingham Study. Stroke. 1991; 22: 312–8.

31. Wannamethee SG, Shaper AG, Whincup PH, Walker M. Smoking cessation and the risk of stroke in middle-aged men. JAMA. 1995; 274: 155–60.

32. Bonita R, Duncan J, Truelsen T, ackson RT, Beaglehhole R. Passive smoking as well as active smoking increases the risk of acute stroke. Tob Control. 1999; 8:156–60.

33. Baillie GM, Sherer JT, Weart CW. Insulin and coronary artery disease: is syndrome X the unifying hypothesis? Ann Pharmacother. 1998; 32: 233–47.

34. Garvey WT, Hermayer KL. Clinical implications of the insulin resistance syndrome. Clin Cornerstone. 1998; 1: 13–28.

35. Kannel WB, McGee DL. Diabetes and cardiovascular disease: the Framingham Study. JAMA. 1979; 241: 2035–8.

36. Tuomilehto J, Rastenyte D. Diabetes and glucose intolerance as risk factors for stroke. J Cardiovasc Risk. 1999; 6: 241–9.

37. Wolf PA, Abbott RD, Kannel WB. Atrial fibrillation as an independent risk factor for stroke: the Framingham Study. Stroke. 1991; 22: 983–8.

38. Benjamin EJ, Wolf PA, D'Agostino RB, Silbershatz H, Kannel WB, Levy D. Impact of atrial fibrillation on the risk of death: the Framingham Heart Study. Circulation. 1998; 98: 946–52.

39. Laupacis A, Albers G, Dalen J. Antithrombotic therapy in atrial fibrillation. Chest. 1998; 114: 579-89.

40. The Atrial Fibrillation Investigators. Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation: analysis of pooled data from five randomized controlled trials. Arch Intern Med. 1994; 154: 1449–57.

41. Di Pasquale G, Urbinati S, Pinelli G. Cardiac investigation in patients with cerebrovascular disease. In: Ginsberg M, Bogousslavsky J. Cerebrovascular Disease: Pathophysiology, Diagnosis and Management. Malden, Mass: Blackwell Science; 1998.

42. Rokey R, Rolak LA, Harati Y, Kutka N, Verani MS. Coronary artery disease in patients with cerebrovascular disease: a prospective study. Ann Neurol. 1984; 16: 50–3.

43. Hertzer NR, Lees CD. Fatal myocardial infarction following carotid endarterectomy: three hundred thirty-five patients followed 6–11 years after operation. Ann Surg. 1981; 194: 212–8.

44. Chimowitz MI, Poole RM, Starling MR, Schwaiger M, Gross MD. Frequency and severity of asymptomatic coronary disease in patients with different causes of stroke. Stroke. 1997; 28: 941–5.

45. The TIMI IIIb Investigators. Effects of tissue plasminogen activator and a comparison of early invasive and conservative strategies in unstable angina and non–Q-wave myocardial infarction: results of the TIMI IIIb trial: Thrombolysis In Myocardial Ischemia. Circulation. 1994; 89: 1545–56.

46. Ohene-Frempong K, Weiner SJ, Sleeper LA, Miller ST, Embury S, Moohr JW et al. Cerebrovascular accidents in sickle cell disease: rates and risk factors. Blood. 1998; 91: 288–94.

47. Adams R, McKie V, Nichols F, Carl E, Zhang DL, Mckie K et al. The use of transcranial ultrasonography to predict stroke in sickle cell disease. N Engl J Med. 1992; 326: 605–10.

48. Ueshima H, Iida M, Shimamoto T, Konishi M, Tsujioka K, Tanigaki M et al. Multivariate analysis of risk factors for stroke: eight-year follow-up study of farming villages in Akita, Japan. Prev Med. 1980; 9: 722–40.

49. Benfante R, Yano K, Hwang LJ, Curb JD, Kagan A, Ross W. Elevated serum cholesterol is a risk factor for both coronary heart disease and thromboembolic stroke in Hawaiian Japanese men: implications of shared risk. Stroke. 1994; 25: 814–20.

50. Iso H, Jacobs DR Jr, Wentworth D, Neaton JD, Conen JD. Serum cholesterol levels and six-year mortality from stroke in 350,977 men screened for the Multiple Risk Factor Intervention Trial. N Engl J Med. 1989; 320: 904-10.

51. Salonen R, Seppanen K, Rauramaa R, Salonen JT. Prevalence of carotid atherosclerosis and serum cholesterol levels in eastern Finland. Atherosclerosis. 1988; 8: 788–92.

52. Dempsey RJ, Diana AL, Moore RW. Thickness of carotid artery atherosclerotic plaque and ischemic risk. Neurosurgery. 1990; 27: 343–8.

53. Atkins D, Psaty BM, Koepsell TD, Longstreth WT Jr, Larson EB. Cholesterol reduction and the risk for stroke in men: a meta-analysis of randomized, controlled trials. Ann Intern Med. 1993; 119: 136–45.

54. Rosenson RS, Tangney CC. Anti atherothrombotic properties of statins: implications for cardiovascular event reduction. JAMA. 1998; 279: 1643–50.

55. Walker SP, Rimm EB, Ascherio A, Kawachi I, Stamfer MJ, Willett WC. Body size and fat distribution as predictors of stroke among US men. Am J Epidemiol. 1996; 144: 1143–50.

56. Rexrode KM, Hennekens CH, Willett WC, Colditz GA, Stamfer MJ, Rich-Edwards JW et al. A prospective study of body mass index, weight change and risk of stroke in women. JAMA. 1997; 277: 1539–45.

57. Abbott RD, Rodriguez BL, Burchfiel CM, Curb JD. Physical activity in older middle-aged men and reduced risk of stroke: the Honolulu Heart Program. Am J Epidemiol. 1994; 139: 881–93.

58. Manson JE, Stampfer MJ, Willett WC. Physical activity and incidence of coronary heart disease and stroke in women. Circulation. 1995; 91:5.

59. Gillum RF, Mussolino ME, Ingram DD. Physical activity and stroke incidence in women and men: the NHANES I Epidemiologic Follow-up Study. Am J Epidemiol. 1996; 143: 860–9.

60. Lakka TA, Salonen JT. Moderate to high intensity conditioning leisure time physical activity and high cardiorespiratory fitness are associated with reduced plasma fibrinogen in eastern Finnish men. J Clin Epidemiol. 1993; 46: 1119-27.

61. Physical activity and cardiovascular health: NIH Consensus Development Panel on Physical Activity and Cardiovascular Health. JAMA. 1996; 276: 241–6.

62. Donahue RP, Abbott RD, Reed DM, Yano K. Alcohol and hemorrhagic stroke: the Honolulu Heart Program. JAMA. 1986; 255: 2311–4.

63. Eikelboom JW, Lonn E, Genest J Jr, Hankey G, Yusuf S. Homocyst(e)ine and cardiovascular disease: a critical review of the epidemiologic evidence. Ann Intern Med. 1999; 131: 363–75.

64. Homocysteine Lowering Trialists' Collaboration. Lowering blood homocysteine with folic acid based supplements: meta-analysis of randomised trials. BMJ. 1998; 316: 894–8.

65. Kaku DA, Lowenstein DH. Emergence of recreational drug abuse as a major risk factor for stroke in young adults. Ann Intern Med. 1990; 113: 821–7.

66. Bushnell CD, Goldstein LB. Ischemic stroke: recognizing risks unique to women. Womens Health Primary Care. 1999; 2: 788–804.

67. Hannaford PC, Croft PR, Kay CR. Oral contraception and stroke: evidence from the Royal College of General Practitioners' Oral Contraception Study. Stroke. 1994; 25: 935–42.

68. Heinemann LA, Lewis MA, Thorogood M, Spitzer WO, Holzmann IG, Brappacher R. Case-control study of oral contraceptives and risk of thromboembolic stroke: results from International Study on Oral Contraceptives and Health of Young Women. BMJ. 1997; 315: 1502–4.

69. Poulter NR, Chang CL, Farley TMM. Ischaemic stroke and combined oral contraceptives: results of an international, multicentre, case-control study: WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. Lancet. 1996; 348: 498–505.

70. Central Bureau of Health Intelligence India. Mortality statistics in India 2006-Status of mortality statistics reporting in India. A Report March 2007. Government of India.

71. Joshi R. Cardona M, Iyengar S, Sukumar A, Raju CR, Raju K et al. Chronic disease now a leading cause of delath in rural India-mortality data from the Andhra Pradesh rural health initiative. Intern J of Epi. 2006; 35: 1522-9.

72. Sethi P, Anand I, Ranjan R, Sethi N, Torgovnick J. Stoke: the neglected epidemic: an Indian perspective. Internet J of Neur. 2007; 8(1): 1-8.

73. Shah B, Mathur P. Workshop Report on Stroke Surveillance in India. Division of Noncommunicable Diseases. New Delhi: Indian Council of Medical Research; 2006.

74. Sridharan S, Unnikrishnan JP, Sukumaran S, Sylaria PN, Nayak SD, Sarma PS . Incidence, types, risk factors and outcome of stroke in a developing country: the Trivadrum Stroke Registry. Stroke 2009; 40: 1212-18

75. Fernandez Jose EF, Martinoz-sanchez M. Report on data collection for smoking and related relative risks. catalan institute of oncology: 2010 Sep. 26.

76. Aiyar. A Study of clinic-radiological correlation in cerebrovascular stroke. Gujarat Medical Journal. 1999 Mar. 52,

77. Chitrambalam P, Baskar D, Revathy S. A study on stroke in young and elderly in Rajiv Gandhi government general hospital, Chennai. Int J Clin Med. 2012; 3: 184-9.

78. Gauri LA, Kochar DK, Joshi A, Jain R, Gupta S, Saini G et al. A study of risk factors & clinical profile of stroke at Bikaner. J API. 2000 Jan; 48(1).

79. Naik M, Rauniyar RK, Sharma UK, Dwevedi S, Karki DB, Samual JR. Clinic-radiological profile of stroke in eastern Nepal: A computed tomographic study. Kathmandu University of medical council. 2006; 4(14): 161-6.

80. Pinhero L, Damodar S, Roy AK. Risk factors in stroke:A prospective study. JAPI. 2000 Jan; 48(1).

81. Eapen RP, Parikh JH, Patel NT. A Study of clinical profile and risk factors in cerebrovascular stroke. Gujarat Medical Journal. 2009 Aug; 64(2).

82. Charles W. Disorders of cerebral circulation. In: Walton John, ed. Brain'sDiseases of The Nervous system, 10th edition. Oxford: Oxford University Press; 2000: 197-265.

83. Benerjee TK, Mukharjee CS, Sarkhel A. Stroke in urban population of Calcutta–An epidemiological study. Neuroepidemiology 2001: 2(3): 201-7.

84. Gresham GE, Fitzpatric TE, Wolf PA. Residual disability in survivors of stroke-The Framingham study. N Engl J Med. 1975: 293: 454-6.

85. Tan KS, Navarro JC, Wong KS, Huang YN, Chiu HC, Poungvarin N et al. Clinical profile, risk factors and aetiology of young ischaemic stroke patients in Asia: A prospective multicentre, observational study in eight cities: Neurology Asia. 2014; 19(2): 117-27.

86. Chapman JM, Reeder LG, Borun ER, Clark VA, Coulson AH. Epidemiology of vascular lesions affecting the central nervous system: the occurrence of strokes in a sample population under observation for cardiovascular disease. Am J Public Health. 1966; 56: 191.

87. Srinivasan K. Ischemic cerebrovascular disease in the young. Two common causes in India. Stroke. 1984; 15: 733-5.

88. Donnan GA, Mcneil JJ, Adena MA. Smoking as a risk factor for cerebral ischaemia. Lancet. 1989; 8664: 643-7.

89. Kaul S, Venketswamy P, Meena AK, Sahay R, Murthy JM. Frequency, clinical features and risk factors of lacunar infarction (data from a stroke registry in south India). Neurology india. 2000 Jun; 48(2): 116-9.

90. Fletcher GF. Exercise in the prevention of stroke. Health Rep. 1994; 6: 106–10.

91. Lakka TA, Salonen JT. Moderate to high intensity conditioning leisure time physical activity and high cardiorespiratory fitness are associated with reduced plasma fibrinogen in eastern Finnish men. J Clin Epidemiol. 1993; 46: 1119–27.

92. Ukoha OB, Ajaegbu O, Eke CO. A review of stroke cases in a military hospital in Nigeria. AFRIMEDIC J. 2012 July-Dec; 3(2): 30-3.

93. Sallam AR, Al-Aghbari K. The clinical profile of stroke: a Yemeni experience. J Med J. 2009; 43(2): 115-21.

94. Devichand, Caroli RK. A study of cerebrovascular strokes: J Indian Med.Asso. 1991 Jan; 36(12).

PROFORMA

NAME :

AGE/SEX :

IP NO :

OCCUPATION :

ADDRESS :

FAMILY INCOME :

PRESENTING COMPLAINTS :

	YES	NO
WEAKNESS		
HEADACHE		
VOMITING		
LOSS OF CONSCIOUS		
SEIZURES		
UNSTEADY GAIT		
SPEECH DISTURBANCES		

ONSET OF SYMPTOMS :

SUDDEN GRADUAL

PAST HISTORY :

- SYSTEMIC HYPERTENSION
- DIABETES MELLITUS
- PREVIOUS HISTORY OF STROKE OR TIA
- PREVIOUS RHEUMATIC HEART DISEASE/ CORONARY ARTERY DISEASE /ATRIAL FIBRILLATION
- SMOKING
- ALCOHOLISM

FAMILY HISTORY :

CLINICAL EXAMINATION :

Height :

Weight :

BMI :

BP :

TEMPERATURE :

CARDIOVASCULAR SYSTEM :

RESPIRATORY SYSTEM :

ABDOMEN :

CNS :

LAB INVESTIGATIONS :

RBS :

FBS / PPBS (IF NEEDED) :

URINE SUGAR :

LIPID PROFILE :

ECG :

ECHOCARDIOGRAM :

CT BRAIN / MRI BRAIN :

a) INFRACTION B) HAEMORRHAGIC TRANSFORMATION

VESSEL INVOLVEMENT

MCA

ACA

MASTER CHART

S.No.	NAME	AGE	SEX	MOTOR	SPEECH	ALTERTED SENSORIUM	CRANIAL NERVE	DIABETES	SHT	DYSLIPIDEMIA	OBESITY	SMOKING	ALCOHOL	HEART DISEASE \AF	PAST H\O STROKE OR TIA	FAMILY H\O	Socio Economic Status (Prasad Classification)
1	PALANI	50	M	Lt	NO	YES	L7U	YES	NO	NO	NO	YES	YES	NO	NO	NO	class 3
2	RAJAN	56	M	Lt	D	NO	L7U	YES	NO	NO	NO	YES	NO	YES	NO	NO	class 5
3	JOTHY	60	M	Rt	NO	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 4
4	GANAPATHI	62	M	Rt	D	NO	R7U	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 5
5	KRISHNAN	66	M	Rt	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 4
6	KUPUSWAMY M	64	M	NO	D	NO	R7U	NO	YES	NO	NO	NO	NO	YES	NO	NO	class 5
7	SADHIQBASHA	69	M	Rt	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 4
8	MUNIYANDI	49	M	Rt	D	NO	R7U	YES	YES	NO	NO	NO	NO	NO	NO	NO	class 5
9	GOPALAN	55	M	NO	NO	YES	NO	NO	YES	YES	NO	NO	NO	NO	YES	NO	class 3
10	PARTHIBAN	53	M	Rt	NO	NO	R7U	YES	YES	NO	NO	YES	YES	NO	NO	NO	class 3
11	MUNUSWAMY	77	M	Rt	NO	NO	R7U	NO	YES	YES	YES	NO	NO	NO	NO	NO	class 5
12	KUPPUSAMY N	72	M	Lt	D	NO	L7U	NO	YES	NO	NO	NO	NO	YES	NO	NO	class 5
13	ANANTHAN	52	M	Rt	NO	NO	R7U	NO	YES	NO	NO	YES	YES	NO	NO	NO	class 5
14	ARUMUGAM	78	M	Rt	NO	NO	R7U	YES	NO	YES	NO	YES	NO	NO	NO	NO	class 4
15	MUNIYAPPAN	67	M	Rt	NO	YES	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	class 5
16	LAKSHMI	49	F	Rt	D	NO	R7U	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 4
17	THEIVANAI	71	F	Lt	NO	NO	L7U	NO	YES	NO	NO	NO	NO	NO	YES	NO	class 5
18	SARASWATHY	66	F	Lt	D	NO	L7U	NO	YES	YES	NO	NO	NO	NO	NO	NO	class 3

S.No.	NAME	AGE	SEX	MOTOR	SPEECH	ALTERTED SENSORIUM	CRANIAL NERVE	DIABETES	SHT	DYSLIPIDEMIA	OBESITY	SMOKING	ALCOHOL	HEART DISEASE \AF	PAST H\O STROKE OR TIA	FAMILY H\O	Socio Economic Status (Prasad Classification)
19	MANIKAM	48	M	Rt	NO	NO	R7U	NO	YES	YES	YES	NO	NO	NO	NO	NO	class 5
20	KRISHNAVENI	65	F	NO	D	YES	L7U	YES	NO	NO	NO	NO	NO	YES	NO	NO	class 3
21	MANIKAVEL	49	M	Rt	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	YES	class 5
22	FAIZAL	79	M	Lt	D	NO	L7U	NO	YES	NO	NO	YES	YES	NO	NO	NO	class 4
23	PARVEEN	38	F	Rt	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 4
24	KUPPAN	65	M	Lt	NO	NO	L7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
25	KRISHNAMORTHI	62	M	Rt	A(m)	NO	R7U	NO	YES	YES	YES	YES	YES	NO	YES	NO	class 4
26	RAMAN	39	M	Rt	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
27	KANTHAN	21	M	Rt	NO	NO	R7U	YES	NO	NO	NO	NO	NO	NO	NO	NO	class 3
28	KARUPAN	54	M	Lt	D	NO	L7U	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 5
29	PARVATHI	65	F	Lt	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 4
30	GOPALSAMY	56	M	Lt	D	NO	L7U	NO	YES	NO	NO	YES	YES	NO	NO	NO	class 5
31	PONNAIYAN	67	M	Rt	NO	NO	R7U	NO	YES	NO	NO	YES	NO	YES	NO	NO	class 2
32	MURUGESAN	78	M	Lt	D	NO	L7U	NO	YES	NO	NO	YES	YES	NO	NO	NO	class 4
33	BALAMURUGAN	77	M	Lt	D	NO	L7U	YES	YES	NO	NO	YES	NO	YES	NO	NO	class 3
34	MUNUSWAMY	54	M	Lt	NO	NO	NO	NO	YES	NO	NO	NO	YES	NO	NO	NO	class 4
35	MUTHUSAMY	40	M	Rt	D	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
36	TIRUPATHI	24	M	Rt	NO	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 3
37	RAMESH	56	M	Lt	D	YES	R6,7,12	NO	YES	NO	NO	YES	YES	NO	NO	NO	class 5
38	RANJITH	58	M	Lt	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	class 4

S.No.	NAME	AGE	SEX	MOTOR	SPEECH	ALTERTED SENSORIUM	CRANIAL NERVE	DIABETES	SHT	DYSLIPIDEMIA	OBESITY	SMOKING	ALCOHOL	HEART DISEASE \AF	PAST H\O STROKE OR TIA	FAMILY H\O	Socio Economic Status (Prasad Classification)
39	KUPAMMAL	66	F	NO	NO	NO	R7U	NO	YES	NO	NO	NO	NO	YES	YES	NO	class 4
40	MANIVANNAN	40	M	Rt	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	class 5
41	CHANDRAN	42	M	Lt	D	NO	L7U	NO	YES	NO	NO	NO	YES	NO	NO	NO	class 4
42	MANIKANDAN	67	M	Rt	A(g)	YES	R7U	YES	NO	YES	YES	YES	YES	NO	NO	NO	class 5
43	ANDIYAPPAN	78	M	Lt	D	NO	L7U	NO	YES	YES	YES	YES	NO	NO	YES	NO	class 5
44	SHIVAN	47	M	Lt	NO	NO	L7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
45	RAJENDRAN	54	M	Rt	A(g)	YES	R7U	YES	YES	NO	NO	YES	YES	YES	NO	NO	class 5
46	PANDIYAN	53	M	Lt	NO	NO	NO	NO	YES	NO	NO	YES	YES	NO	YES	NO	class 5
47	LOGAMMAL	47	F	Lt	D	NO	L7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
48	KANNAN	61	M	Rt	D	NO	R7U	NO	YES	NO	NO	NO	YES	NO	NO	NO	class 5
49	KATHIRVEL	69	M	Rt	NO	NO	L 3	NO	YES	NO	NO	YES	YES	NO	NO	NO	class 3
50	MUTHUPANDI	52	M	Lt	NO	NO	L7U	NO	NO	NO	NO	YES	NO	NO	NO	NO	class 5
51	CHELLAPAN	67	M	Lt	D	NO	L7U	NO	YES	NO	NO	YES	YES	NO	NO	NO	class 5
52	MAARIMUTHU	75	M	Rt	D	YES	R7U	NO	YES	NO	NO	NO	YES	NO	NO	NO	class 5
53	CHINNADURAI	53	M	Lt	NO	NO	L7U	YES	NO	YES	NO	NO	YES	NO	NO	NO	class 4
54	DURAISWAMY	39	M	Rt	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
55	PERIYAKARUPU	69	M	Lt	NO	NO	L7U	NO	YES	NO	NO	NO	YES	YES	NO	NO	class 3
56	THAKOOR BASHA	75	M	Rt	D	NO	R7U	NO	NO	YES	YES	YES	NO	NO	NO	YES	class 5
57	JOSEPH	54	M	Lt	D	YES	L7U	YES	NO	YES	YES	NO	YES	NO	NO	NO	class 3
58	RAMAN	67	M	NO	NO	NO	9 ,10	YES	YES	NO	NO	YES	YES	NO	NO	NO	class 5

S.No.	NAME	AGE	SEX	MOTOR	SPEECH	ALTERTED SENSORIUM	CRANIAL NERVE	DIABETES	SHT	DYSLIPIDEMIA	OBESITY	SMOKING	ALCOHOL	HEART DISEASE \AF	PAST H\O STROKE OR TIA	FAMILY H\O	Socio Economic Status (Prasad Classification)
59	SURESH	31	M	Rt	D	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
60	DHANALAKSHMI	74	F	Lt	NO	NO	L7U	NO	YES	YES	NO	NO	NO	NO	NO	NO	class 5
61	SAMPATH	67	M	Lt	D	NO	L7U	NO	NO	NO	NO	YES	NO	NO	NO	NO	class 5
62	JOHN PANDIYAN	69	M	Lt	NO	NO	NO	YES	YES	NO	NO	YES	YES	NO	YES	NO	class 5
63	SYED AHMED	33	M	Rt	D	NO	R7U	YES	NO	NO	NO	NO	NO	NO	NO	NO	class 5
64	VEERAMMAL	34	F	Rt	D	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 4
65	PERIYASWAMY	66	M	Lt	NO	NO	L7U	NO	YES	NO	NO	NO	YES	NO	NO	NO	class 5
66	CHINNAPONNU	54	F	Lt	NO	NO	L7U	NO	YES	YES	YES	NO	NO	YES	NO	NO	class 5
67	BALAIYA	77	M	Rt	NO	NO	NO	YES	YES	NO	NO	YES	YES	NO	NO	NO	class 5
68	BHARATHIRAJA	56	M	NO	NO	YES	R 3	NO	YES	NO	NO	YES	YES	NO	NO	NO	class 3
69	RANI	49	F	Lt	NO	NO	L7U	NO	NO	YES	YES	NO	NO	NO	NO	NO	class 5
70	NEDUMARAN	47	M	Rt	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	class 5
71	DHANDAPANI	56	M	Lt	NO	NO	L7U	NO	YES	NO	NO	YES	YES	YES	NO	NO	class 5
72	MANIVELAN	68	M	Rt	A(m)	NO	NO	NO	NO	YES	YES	YES	YES	NO	YES	NO	class 5
73	MARUTHAPANDI	25	M	Rt	D	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
74	VIJAYKUMAR	40	M	Lt	D	NO	L7U	NO	YES	NO	NO	NO	YES	NO	NO	NO	class 5
75	BALAKRISHNAN	65	M	Rt	NO	NO	NO	NO	YES	YES	YES	NO	YES	NO	NO	NO	class 5
76	MUTHAYAMMAL	68	F	Lt	D	NO	L7U	YES	YES	NO	NO	NO	NO	NO	NO	NO	class 5
77	KRISHNAVENI	78	F	NO	NO	YES	NO	NO	YES	YES	YES	NO	NO	YES	NO	NO	class 5

S.No.	NAME	AGE	SEX	MOTOR	SPEECH	ALTERTED SENSORIUM	CRANIAL NERVE	DIABETES	SHT	DYSLIPIDEMIA	OBESITY	SMOKING	ALCOHOL	HEART DISEASE \AF	PAST H\O STROKE OR TIA	FAMILY H\O	Socio Economic Status (Prasad Classification)
78	MALATHI	54	F	Lt	NO	NO	L7U	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 4
79	RAJARAJAN	59	M	Lt	NO	NO	NO	NO	YES	NO	NO	YES	YES	NO	YES	NO	class 5
80	VIJAYALAKSHMI	60	F	Lt	D	NO	L7U	NO	YES	YES	NO	NO	NO	YES	NO	NO	class 5
81	KUPAMMAL	27	F	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
82	VELU	42	M	Lt	NO	NO	L7U	NO	YES	NO	NO	YES	NO	NO	NO	NO	class 5
83	ANNAMALAI	40	M	Rt	D	NO	R7U	NO	NO	NO	NO	NO	YES	NO	NO	NO	class 4
84	STEPHAN	57	M	Lt	NO	NO	L7U	NO	YES	NO	NO	YES	YES	YES	YES	NO	class 5
85	MARY	70	F	Lt	NO	NO	L7U	YES	YES	NO	NO	NO	NO	NO	NO	NO	class 5
86	VASANTHI	65	F	Lt	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 5
87	MOORTHY	56	M	Rt	A(m)	NO	R7U	YES	YES	NO	NO	YES	YES	NO	NO	NO	class 5
88	VIJAYALAKSHMI	76	F	Rt	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 5
89	MAARIAMMA	78	F	NO	D	NO	L7U	YES	YES	NO	NO	NO	NO	NO	NO	NO	class 5
90	SOMASUNDARAM	60	M	Lt	NO	NO	L7U	YES	YES	NO	NO	YES	NO	NO	NO	NO	class 5
91	ELUMALAI	54	M	Lt	NO	NO	NO	YES	YES	NO	NO	YES	YES	NO	NO	NO	class 4
92	AHAMED BASHA	40	M	Rt	D	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 3
93	BALU	31	M	Rt	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
94	SELVARAJ	26	M	NO	A(m)	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
95	AROKIAMARY	67	F	Lt	NO	NO	L7U	NO	YES	NO	NO	NO	NO	NO	NO	NO	class 5
96	PAATTAPAN	50	M	Lt	D	NO	L7U	YES	YES	NO	NO	YES	NO	NO	NO	NO	class 4
97	JOTHILAKSHMI	22	F	Rt	D	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5

S.No.	NAME	AGE	SEX	MOTOR	SPEECH	ALTERTED SENSORIUM	CRANIAL NERVE	DIABETES	SHT	DYSLIPIDEMIA	OBESITY	SMOKING	ALCOHOL	HEART DISEASE \AF	PAST H\O STROKE OR TIA	FAMILY H\O	Socio Economic Status (Prasad Classification)
98	MURUGESAN	48	M	Lt	D	YES	L7U	NO	YES	NO	NO	YES	NO	NO	NO	NO	class 5
99	VEERAMANI	29	M	Rt	NO	NO	R7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5
100	SEVI	32	F	Lt	D	NO	L7U	NO	NO	NO	NO	NO	NO	NO	NO	NO	class 5

KEY TO MASTER CHART

M - MALE

F - FEMALE

RT - RIGHT

LT - LEFT

D - DYSARTHRIA

A (g) - GLOBAL APHASIA

A(m) - MOTOR APHASIA

U - UMN PALSY

L - LMN PALSY

SHT - SYSTEMIC HYPERTENSION

TIA - TRANSIENT ISCHEMIC ATTACK

பங்கேற்பவர்களுக்கு ஆய்வின் விவரம்

ஆய்வின் நோக்கம் :

மூளை ரத்தக் குழாய் அடைப்பினால் வரும் பக்கவாத நோயாளிகளின் மருத்துவ சுயவிவரங்கள் மற்றும் ஆபத்து காரணிகள் குறித்து பின்னோக்கி ஆய்வு செய்தல் .

ஆய்வில் பங்கேற்க தகுதிகள் :

18 வயது முதல் 65 வயது வரையிலான CT/MRI SCAN மூலம் மூளை ரத்தக் குழாய் அடைப்பு உறுதி செய்யப்பட்ட உள்நோயாளிகள்.

செய்முறை விளக்கம் :

இந்த ஆய்வில் பங்கேற்க்கும் நோயாளிகளிடம் இருந்து பக்கவாத நோயின் அறிகுறிகள் மற்றும் நோய்க்கான ஆபத்து காரணிகள் குறித்து சுய விவரங்கள் சேகரிக்கப்படும்.

மருத்துவமனையில் உள்நோயாளியாக அனுமதிக்கப்படுபவர்களுக்கு செய்யப்படும் வழக்கமான ரத்த பரிசோதனை, சிறு நீர் பரிசோதனை, ECG, ECHO மற்றும் CT/MRI SCAN குறித்த விவரங்கள் நோயாளிகளிடம் இருந்து பெறப்படும்.

ஏன் பங்கேற்க வேண்டும் ?

மூளை ரத்தக் குழாய் அடைப்பு நோயாளிகளின் சுய விவரங்கள் மற்றும் ஆபத்து காரணிகளை ஆய்வு செய்வதன் மூலமாக, வாழ்க்கை முறை மாற்றம் மற்றும் ஆபத்துக் காரணிகளுக்கு முன்கூட்டிய சிகிச்சை அளிப்பதன் மூலமாக , சமுதாயத்தில் பக்கவாத நோயின் நிகழ்வை குறைக்க முடியும்.

ஆராய்ச்சி நிலையம் :

பொது மருத்துவத்துறை

அரசு வேலூர் மருத்துவக்கல்லூரி மருத்துவமனை

வேலூர்.

சுய ஒப்புதல் படிவம்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொண்டேன். நான் ஆய்வில் இருந்து விலகி கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்தி கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் ..... இடம் .....

நாள் .....

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம் ..... இடம் .....

நாள் ..... ஆய்வாளரின் பெயர் .....